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MORBI NEURALES

AN ATTEMPT TO APPLY A KEY PRINCIPLE TO THE DIFFERENTIATION OF THE MAJOR GROUPS *

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This paper is intended to be more modest than even its subtitle "attempt" denotes. The writers feel confident that a key principle, roughly reminding one of the principle of Gray's botany for the diagnosis of plants, is applicable to the diagnosis of nervous diseases. Central in the application of such a key principle is the idea of exclusion of diseases in diagnostic consideration by means of following a certain order. But granting the theoretical virtues of a key principle and of the method of orderly diagnosis by exclusion, we must remain uncertain whether the order adopted is the *one best way*, in the sense of the efficiency engineer who devised that term.

The attempt to apply a key principle to the differentiation of the major groups of nervous diseases was based on the comparative success of the application of an identical principle to the differentiation of the major groups of mental diseases. Since the publication of a key to the practical grouping of mental diseases, presented to the American Neurological Association in 1917, the principle of diagnosis per exclusionem in ordine has been presented to the Association of American Physicians in 1918, and in briefer form in a second paper on certain applications of the pragmatic method to psychiatry (1918). The idea of a possible extension of this principle of orderly exclusion to the problems of general medicine has met with the approval of Dr. Richard C. Cabot, who, at the June, 1919, meeting of the Association of American Physicians, displayed a tentative list (without definitive sequence) of the major groups of diseases in general. This list, as the sequel will show, may have some value for clinical neurology, and may even prove superior to the key to mental diseases formerly laid down.

* Read by title at the annual meeting of the American Neurological Association, held at Atlantic City, N. J., June 16-18, 1919.

DIAGNOSIS BY EXCLUSION

Perhaps it is superfluous to argue here for the virtues of the key principle in the form of diagnosis per exclusionem in ordine. In a previous paper considerable argument has been devoted to the habitual underestimation of the values of the method of diagnosis by exclusion, of which DaCosta and his followers were guilty in the sixties and seventies of the last century. The method of diagnosis by exclusion was to be resorted to only in extremis, according to these authors, and especially according to the book on diagnosis by Da Costa, published in 1864, which was thought by some to have marked an epoch in the progress of internal medicine. The so-called indirect diagnosis or diagnosis by exclusion was, according to DaCosta, "not on ordinary occasions much employed, nor indeed," DaCosta continues, "is it to be recommended." DaCosta thought that to prove what a thing is by proving all that it is not is a very tedious process. He went on to say that it was "difficult to think of all the possibilities" and remarked that "pathology was in an imperfect state." But its imperfections seem to us to interfere with all other methods of diagnosis as much as with diagnosis by exclusion. As for the difficulty of thinking of all the possibilities, the device of placing these possibilities in manageable groups (after the manner of the diagnostic groups of animals, plants and minerals, that is, after the manner of Linnaeus), DaCosta and his colleagues in the single-volume textbooks of medicine did not conceive of such a process. Apparently, DaCosta felt that in the process of diagnosis by exclusion one would need to exclude theoretically every other disease than the one on which one would finally fix. As for the tediousness of this process of diagnosis, no doubt accurate diagnosis in difficult cases is and must remain extraordinarily tedious, but remains none the less interesting and important.

But diagnosis by exclusion is not diagnosis by exclusion in order. It is one thing to secure a list; it is another thing to make a sequence of the elements in the list. An aggregate does not forthwith throw itself into order, and especially not into that arithmetical arrangement called a "well-ordered" aggregate.

Now as a matter of fact, it seems to us that most workers in difficult cases do actually employ something like what has been described as diagnosis per exclusionem in ordine. In correspondence with some specialists, affirmative replies have been given to questions on this line. Ordinarily, diagnosticians are prouder, perhaps, of making a diagnosis of smallpox by the sense of smell at 100 yards (more or less) than to make a careful, logical diagnosis by exclusion in order. Yet the most expert diagnosticians are reduced to using diagnosis per exclusionem

from time to time. Even DaCosta admitted that the method had to be used on occasion. We hold that a method which has to be resorted to in difficult cases ought to be rationalized so that tyros and inexpert workers may the earlier learn to make correct diagnoses. We hold that the field of mental and nervous diseases is a good field in which to begin, because the majority of cases in these fields are difficult of diagnosis. Especially in the field of mental diseases is it true that there is practically no single pathognomonic symptom in the whole branch. Starting with a slant toward some disease in which a given symptom is characteristic, the tyro can prove from the very best text-book that almost any mental case he has in hand is or might well be a case of the disease he starts to prove it to be. The tyro is only caught up short when the laboratory comes to the rescue with pathognomonic signs. But the tyro with a slant rushes in where the laboratory expert fears to tread. On this account, as there are practically no indicator symptoms in the field of mental disease, the method of diagnosis per exclusionem in ordine was found to have especial value for the mental diseases.

We shall not here rehearse the situation in the mental disease groups. We may persist in pointing out, however, that, if eleven groups of mental disease have been laid down as of pragmatic orderly value in diagnosis, there is no reason why some other worker may not prove that there are twelve or ten instead of eleven groups. Thus one acute observer believes that there should be added to the eleven-group system, as presented, a twelfth group of paranoic cases. Another acute observer suggests that there are really no senile diseases, that properly obtain the name, at least among the mental diseases, and that consequently the senescent-senile group should be deleted from the sequence and its diseases distributed otherwise; for example, among the arteriosclerotic brain diseases, the manic depressive group, etc. Let us insist, too, that not only may the groups be accordeonized, that is, interpolated and extrapolated without destroying the key principle involved, but the order of the groups may likewise be changed, possibly to advantage. Some considerations on this point have been given in the paper above mentioned on applications of the pragmatic method to psychiatry.

CLASSIFICATION OF DISEASES

For those who are willing to consider a rationalization of the method of diagnosis by exclusion in order (a method which we are convinced that every one dealing with complex cases must in some form employ) there is another bogey of greater difficulty. Pathology, as DaCosta might still remark, is in an imperfect state! Why classify

diseases in any definite manner until we know more about the etiology and genesis of diseases? The answer to this objection is that in practical life some account of stock must continually be taken. Next year the very basis of some well-recognized disease of today may be entirely overthrown; even today we may recognize that the etiology or genetic basis of that particular disease is very shaky or rickety. Nevertheless, it remains our duty to make diagnoses on the best basis available; otherwise there will be no common ground of discussion with colleagues and no common ground for practical measures of treatment. Accordingly, the imperfections of present day pathology should not deter us from practical classifications. We must cheerfully admit that these practical classifications may be changed from decade to decade. The pessimist or the worker weary in well-doing, may regret the long string of obsolete and obsolescent classifications that the world provides, but there is no genuine ground for such regret where the classifications have been made in good faith by careful and logical thinkers. Some authors appear to think that there will some day be a single classification of diseases, and every day we see classifications put forward by eager workers which they seem fondly to wish shall replace all other classifications. A glance into logical works on the topic of classification, even into the ancient work of John Stuart Mill called a System of Logic, would reveal to these workers the infinitude of classifications of the very same objects. A classification by causes may totally differ from a classification by genesis, and both may differ from a classification for the purposes of diagnosis, and in the end a classification by methods of treatment may be found to cut quite across all other classifications. Then, too, it is probably worth while to devote a sentence to the universal occurrence of the editorial imbecile (or shall we say idiot?) who makes fun of what he calls the classifying mania, apparently not being aware that almost the whole development of science and art depends on this so-called mania.

The etiologist may have in mind some particular factors which he would like to elevate into the so-called causes of disease. At the other end of the line some hospital administrator will be found who would like to classify all cases according to the amount of food, nursing and open plumbing which they may require. In between these two extremes may be found the practical diagnostician who has troubles of his own. The present effort deals with the troubles of the diagnostician rather than with the troubles of the etiologist or of the hospital administrator.

With more particular respect to the nervous diseases themselves is it not possible that the nervous diseases will very shortly fall into a common group with the mental diseases? Is there any point in producing an order of nervous diseases independently of an order of

mental diseases? Probably the new art of neuropsychiatry is on its way, and neuropsychiatrists with equally good training on both sides will become available. However, we believe that it is the pragmatic situation at the present time that psychiatrists and clinical neurologists are two separate and different kinds of specialists. No doubt the fundamental basis of both arts lies in the science of neuropathology, taken in its widest sense of including both structural and functional concepts—but that wide and deep science is still (to borrow a leaf out of DaCosta's vocabulary) in an imperfect state. The question is a practical one. It is something like the question whether we ought to separate the specialties of dermatology and syphilis or the specialties of surgery and gynecology. Now one opinion, now another, will prevail, and there will be a progress toward a final decision.

Classification of Nervous Diseases.—As we approach the topic of the nervous diseases taken in their general sense, we find that the distinction between "organic" and "functional" nervous diseases is the most fundamental one in many systematic works and in perhaps the majority of nonspecialistic medical minds. Is a case organic or is it functional? — this is about as far as the general practitioner likes to wander in the field of clinical neurology. He has an idea that by means of the "reflexes" the neurologist more or less effectively arrives at a diagnosis of organic nervous disease and is often somewhat astonished that the neurologist cannot, by doubling or trebling the number of "reflexes" taken, somehow arrive at a diagnosis doubly or trebly accurate. In our view, this no doubt perfectly true distinction between organic and functional diseases is of little diagnostic value in the individual case, and this is aside from any doubts we may have as to the definition of the term "functional."

We examined the nervous diseases in sundry systematic works and seemed to be able to put these diseases into five groups as follows:

Tentative sequence of major groups of nervous diseases due to

1. Infection.
2. Historrhexis.
3. Neuronatrophy.
4. Imbalance.
5. Special.

Application of Classification in Diagnosis by Exclusion.—Another word as to its application by the tyro or by the general practitioner. Let us suppose the general practitioner confronted by a paralysis. Suppose in the course of his thinking he lights on the hypothesis that it is due to "neuritis." He may then consider or look up in the books

all the types of neuritis which he can find and look for signs or symptoms appropriate to one or the other of these groups. Now, of course, the diagnosis of neuritis may in itself be erroneous to start with, and the inquiry as initiated may become useless. According to our ideas, it would be much more to the advantage of the patient if the diagnostician proceed somewhat as follows: Here we have a case of paralysis of a limb! In the first place, is this paralysis at all related to infection? Are there any signs of (1) infection whether in temperature, in blood, or otherwise, in this patient? What tests should be employed to secure evidence on the point? In the second place, if the case shows no sign of infection let us turn to the question of injury or other lesion of whatever sort which may have affected the tissues from without. Even if we cannot absolutely exclude infection as a matter of fact, let us for the purposes of diagnosis exclude it as a matter of argument until the rest of our tests have become available. Excluding infection, is there any evidence in the situation of a condition to which we above gave the name (2) historrhexis? Historrhexis might include the severing of a nerve or the nerve tissues by a knife or a bullet. It would include the blocking of nerves and the by-effects of historrhexis. For diagnostic purposes it would include the effects of an apoplexy not only as directly due to the destruction of brain tissue, but also to the pressure on collateral tissues. Not only trauma and arteriosclerosis, but also tumors, would be included in this concept of historrhexis, from which for our purposes the orderly exclusion would have already excluded infection. The historrhexis inquiry sums up to an inquiry for trauma or other tissue-injuring lesions of noninfectious nature and of a focal character.

Thirdly, the diagnostician, having got rid of the hypothesis of (1) infection (including, of course, neurosyphilis) and of trauma and other forms of (2) historrhexis, would arrive, according to our scheme, to the group which we have termed (3) the neuronatrophies. Our first thought was to name these the "scleroses," since the group contains the various classic scleroses, like lateral sclerosis, multiple sclerosis, etc. But, on the whole, the primary fact appears to be that of neuronatrophy, a condition not of the general nature of historrhexis, but of a much finer and more differentiated type. With the infections and the common historrhexes out of the diagnostic way, the tyro would now come to the neuronatrophies, that is, to questions of combined system disease, syringomyelia, multiple sclerosis, etc. (The fact that, e. g., syringomyelia often results in historrhexis with secondary degeneration, should not bother the diagnostician a whit. He has already considered the historrhexes and eliminated perhaps a transverse myelitis from the field of diagnosis. He does not arrive primarily at

the question of syringomyelia, but only after having eliminated the effects of historrhexis [pressure myelitis] and infections [Pott's disease] which should be discriminated from syringomyelia.) (Again, some one might prove that multiple sclerosis is infectious. But the infectious group of nervous diseases, excluding some peripheral forms, is essentially a group of diseases in which pleocytosis of the spinal fluid is characteristic diagnostically. Multiple sclerosis appears to be in general outside this group. For the present pragmatic purposes multiple sclerosis should evidently fall among the classic neuronatrophies rather than among the historrhexes or infections of the nervous system.)

Fourthly, if the diagnostician has been able to eliminate (1) infections, (2) rhexes of tissues, and (3) differential atrophies of neuron system, he will arrive at a somewhat nondescript group which we above termed (4) imbalance, under which the drug-neuritis cases, sundry metabolic disorders, the endocrinopathies and sympathetic system disorders fall. A great many of these conditions are conditions of what we might term tissue-imbalance. Imbalance is particularly well shown in the endocrine and in the various sympathetic disorders, and may also be shown in the metabolic group in some sense. Whether multiple neuritis should not sometimes be grouped among the infections and sometimes among metabolic conditions is a question which we do not care to take up. Our purpose is not to add to theories concerning causation or genesis, but merely to take the diseases as they stand in the systematic works and throw them into an effective diagnostic order for the purposes of orderly exclusion.

Fifthly, over and above the (1) infections, (2) historrhexes, (3) neuronatrophies and (4) imbalance groups we have (5) a special or miscellaneous group in the field of nervous diseases, including such conditions as the various aglias, migraines, tics, spasms and vertigo, to say nothing of myopathies, osteopathies, various tissue atrophies, unexplained hydrocephalus, etc. With respect to this last group, the modesty of the clinical neurologists must forbid their complaining about how little they know. The aim of the present classification of nervous diseases is not to display the ego of clinical neurologists, but to find a method for arriving at diagnoses in a more logical manner.

Comments on This System.—We conceive that a system like that of classification into these five groups will serve to clarify many problems in the tyro's mind. The expert may not need it. We suspect that the expert is already using some such method, perhaps one private to himself which he has never thought of betraying to ordinary man. In conversation with various diagnosticians we find that they habitually use such systematic works as Oppenheim in a manner

peculiar to themselves. Diseases in a book like Oppenheim's are worked over precisely as if they were in no important order whatever. We do not deny that Oppenheim's excellent work arranges the diseases in an order on a certain basis. We deny that the order adopted has much to do with practical diagnosis. Practical diagnosis in clinical neurology appears to have been chiefly a topical diagnosis devised to find, in the brain or spinal cord, the seat of lesions chiefly of the group we have above termed historrhexes. To adopt a more general medical attitude such as that defined in these five groups is a plan not found in textbooks of clinical neurology. We suspect, however, that the more mature the clinical neurologists become, the more they arrive at some such practical method for diagnosis by exclusion in order.

We have no idea of insisting that the order we here present is a final order, that it is an order which forbids interpolation or extrapolation, or that it is an order that cannot be changed to advantage. We would like to have the topic considered carefully by those who are concerned, and we are of the opinion that some uniformity of the scheme of elimination for practical diagnostic purposes for the various nervous diseases will greatly benefit the treatment of patients. If, by the naked eye, one has somehow guessed that a patient has a nervous disease, perhaps by means of the low power of the logical microscope, he will come to one of the five groups above mentioned as the logical site of the disease in question. Suppose now that he turns on the somewhat higher powers of the logical microscope. The five groups are subdivided somewhat as follows:

Subdivision of Five Groups of Nervous Diseases

1. Infections
   - Treponema
   - Bacteria
   - Parasites
   - Unknown infections
2. Historrhexes, focal destruction (non-infectious)
   - Trauma
   - Vascular
   - Heightened intracranial pressure
3. Neuronatrophies, scleroses ("classical degenerations")
   - Systemic scleroses
   - Diffuse scleroses
   - Hereditary scleroses
4. Imbalance exo(neuro)genous
   - Drug
   - Metabolic
   - Endocrine
   - Sympathetic
5. Miscellaneous
   - Algias, migraine, vertigo
   - Tics, spasms
   - Myopathy
   - Osteoarthropathy
   - Tissue atrophies
   - Hydrocephalus
   - *Et al.*

SYNOPSIS

1. Infection

(Diagnosis by signs of infection, pleocytosis of liquor, etc.)

Treponema	*Bacteria*	*Unknown*
Tabes	Epidemic meningitis	Poliomyelitis
Pachymeningitis cervicales hypertrophica	Tuberculous meningitis	Hydrophobia
General paresis	Streptococcus meningitis	Landry's disease
	Neuritis	Herpes zoster
	Tetanus	Chorea minor
	Myelitis	
	Encephalomyelitis	
	Abscess	

2. Historrhexis (Focal Destruction)

(Diagnosis by reflex-signs, heightened intracranial pressure, etc.)

Trauma	*Vascular*	*Tumor*
Caisson	Little's disease	Recklinghausen's disease
	Hematomyelia	
	Anemia	
	Hyperemia	
	Hemorrhage	
	Softening	
	Hemorrhagic encephalitis	
	Polioencephalitis, acute hemorrhagic, superior	
	Sinus thrombosis	
	Aneurysm	
	Pseudobulbar palsy?	

3. Neuronatrophy

(Diagnosis by characteristic picture and history)

Systemic Sclerosis	*Diffuse Sclerosis*	*Hereditary Sclerosis*
Lateral sclerosis	Multiple sclerosis	Friedreich's ataxia
Combined system	Pseudosclerosis?	Amaurotic idiocy
Amyotrophic lateral sclerosis	Gliosis spinalis	Familial infantile paralysis
Primary muscular atrophy	Syringomyelia	Primary muscular atrophy, familial form (bulbar form)
Senile paraplegia	Paralysis agitans	Huntington's chorea

4. Imbalance

(Diagnosis by characteristic picture)

Drug *	*Metabolic Disorder*	*Endocrinopathy*	*Sympathetic*
Neuritis	Neuritis	Spasmophilia	Acroparesthesia
Multiple neuritis	Diabetic, senile, etc.	Tetany	Angioneurotic edema
			Myxedema intermittent
		Exophthalmic goiter	Hydrops articulorum
		Cretinism	Raynaud's disease
		Infantilism	Erythromelalgia
		Acromegaly	Scleroderma

* It is possible that these cases should be placed under historrhexis, although many of the phenomena are reversible.

5. Miscellaneous

Algeas	*Migraine*	*Vertigo*	*Tics—Spasms*
			Occupation-neuroses
			Tic convulsive

Myopathy	*Osteoarthropathy*	*Tissue Atrophies*
Primary myopathy	Vertebral disease	Hemiatrophy facialis
Dystrophy, muscular progressive	Luxation—fracture	Hemihypertrophy facialis
Thomsen's disease	Caries	
Myasthenia gravis	Tremors	
Paramyoclonus	Syphilitic	
	Arthritis deformans	
	Osteomalaceous paralysis	

Hydrocephalus	*Undefined*
Meningitis serosa	Caisson
	Anomalies
	Reflex paralysis
	Exhaustion paralysis
	Periodic paralysis

SUMMARY

The method of diagnosis by orderly exclusion, already proposed for use in the diagnosis of mental diseases, is probably of equal value in the field of nervous diseases. The writers have endeavored to gather the main types of nervous disease into a comparatively small number of groups for successive consideration by the tyro, or even by the expert, in diagnostic elimination. Experts may prefer a different order from the one proposed; but it is unlikely that any neurologist fails to use, consciously or unconsciously, some form of orderly diagnosis.

Yet the student is quite likely to be taught that the best procedure is to pick out some striking symptom in a case under consideration, and to follow that symptom back to textbook models for a suggestion as to the entity involved. He then endeavors to match the data of the case in hand with the possibilities laid down in the textbook.

We think that it is much more desirable to take the general situation in the body at large into account, and to include, if possible, first, the hypothesis of an infectious origin for the symptoms. Secondly, we try to exclude, if possible, the effects of coarse and otherwise destructive lesions of the nervous system (historrhexes) namely, in general such conditions as show no signs of infection, but exhibit reflex disorders and signs of heightened intracranial pressure and the like, suggestive of focal lesion. Thirdly, we come to the hypothesis of the existence of one or other of those classic degenerations with which the neurologist is familiar. If infection, historrhexis, and classic degenerations can be excluded, we then proceed, fourthly, to the

hypothesis of some kind of imbalance, perhaps metabolic or endocrine or sympathetic. If the diagnosis cannot be made on these lines, possibly the condition belongs in some miscellaneous and otherwise undefined or highly specialized group.

Even when the disease seems to be limited to a peripheral nerve, we consider that then the successive hypotheses of (1) infection, (2) historrhexis, (3) specialized neuronatrophy, (4) imbalance, can be preferably considered in that order. We think that by the pursuit of some such method as this the neurologist can bring his work better into line with that of general medicine. The method is, moreover, a very pragmatic method, since lines of treatment are specially indicated for the different great groups of disorders. But in so difficult a field we do not wish to dogmatize, and shall be content if our communication arouses interest in the application of the key or order principle to the diagnosis of nervous diseases.

THE FUNCTIONS OF THE CEREBROSPINAL FLUID

WITH A SPECIAL CONSIDERATION OF SPINAL DRAINAGE AND OF INTRASPINAL INJECTIONS OF ARSPHENAMIZED SERUM *

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PHILADELPHIA

THE PROBLEMS PRESENTED

The cerebrospinal fluid has of late years acquired a peculiar importance. This is true not only because of the rôle it has assumed in neurologic diagnosis, but also because of the various attempts that have been made to utilize it as a vehicle of therapeutic administration, or rather, because of the various procedures that have been evolved to use the subarachnoid space and the other containers of the cerebrospinal fluid as avenues of medication. Perhaps it will not be out of place to review some of the facts in regard to its physical and chemical constitution; in regard to its location and distribution; in regard to the laws of physics which it must obey; in regard to its source, it renewal and its escape; in regard to its relation to the vascular and lymphatic systems; in regard to the pressure under which it exists, and, finally, in regard to its movements and other cognate factors. To me it has seemed that the rôle and function of the cerebrospinal fluid has been strangely misinterpreted, and that this has led to attempts at therapeutic administration which may be justly characterized as unscientific; which have not been without danger, and which have either failed of their object or have owed such success as seemed to attend their application, to other and purely incidental factors which have been overlooked; this I hope to show. In any event, a consideration of the cerebrospinal fluid appears to me to be a very timely topic.

SIGNIFICANCE OF THE CHEMICAL COMPOSITION OF THE CEREBROSPINAL FLUID

When we turn our attention to the chemical composition of the cerebrospinal fluid, we are at once impressed by its resemblance to the ordinary three quarter per cent. common salt solution of the histologic laboratory. According to Halliburton,[1] it consists of water, inorganic salts similar to those of the blood plasma, a trace of coagu-

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* Read before the Philadelphia Clinical Association, Dec. 8, 1919.

1. Halliburton, W. D.: The Possible Function of the Cerebrospinal Fluid, Brain, **39**:214 (Oct.) 1916.

lable protein and a certain amount of glucose. It is practically free from formed elements. Halliburton speaks of it as an "ideal physiological salt-solution," "as the perfect physiological medium," more perfect doubtless than the artificial fluids we can make in the laboratory, but in its essential features closely resembling those associated with the names of Ringer and Locke.[2] Mestrezat[3] has pointed out that the cerebrospinal fluid is identical with the ocular and labyrinthine liquids, and he has also emphasized the fact that the purely physical rôle of the latter is altogether indisputable. The liquid of the internal ear communicates directly with the subarachnoid space by means of the aqueduct of the cochlea, and it is proper to infer that it has no other origin; the mediums of the eye are independent, but they are beyond doubt formed by the ciliary processes which appear to be in every way comparable to the choroid plexuses. Mestrezat concludes that the cerebrospinal, the ocular and the labyrinthine fluids form a homogeneous family of liquids possessing a constitution almost exclusively mineral. They are liquids of "remplissage," i. e., of space filling, and of support and protection, and form a group of liquids by themselves. They owe their physiologic properties in part to their perfect fluidity which enables them to penetrate into the smallest interstices. The tonicity of the cerebrospinal fluid, as pointed out by Bard,[4] is constituted almost exclusively by the amount of contained sodium chlorid. This constitutes a negative property, i. e., a property which insures the perfect innocuousness of the liquid to the living elements with which it is in contact.

HYDROSTATIC PROPERTIES

Further, immersed in this salt solution, the brain is supported and buoyed so that its weight is relatively diminished. Mestrezat has calculated that the specific gravity of the cerebrospinal fluid being about 1.0075, and that of the brain 1.032 (women) to 1.041 (men), the pressure which the weight of the brain exerts on its base is reduced to the very low figures of from 26 to 39 grams. From these facts we may infer that a brain weighing from 45 to 50 ounces exerts a pressure of from about three fifths of an ounce to 1¼ of an ounce on its base.

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2. Locke's fluid contains besides water and common salt, minute quantities of potassium chlorid, calcium chlorid, sodium bicarbonate and glucose. See Halliburton, Handbook of Physiology, 14th Ed., London, John Murray, 1919.

3. Mestrezat, William: Le liquide céphalo-rachidien normal et pathologique, Paris, A. Maloine, 1912.

4. Bard, L.: Le rôle de la pression dans l'action physiologique du liqueur céphalo-rachidien. Ses rapports avec les fonctions des plexus choroides et de la glande pinéale, J. de physiol. et de path. gén. **17**:171, 1917.

Further, this pressure is distributed over a relatively wide area. By means of this hydraulic suspension — for such it is — not only do the vessels of the base escape all compression from the weight of the brain, but the brain itself escapes the effects of pressure on its surfaces of support. Flattenings, deformities and distortions are prevented by means of this hydraulic suspension.

This hydraulic suspension and the relative loss of weight of the brain leads to another important result: The force of the impact of the brain on the hard cranial walls is diminished, i. e., the force of the blow which the brain itself, under given conditions, strikes against the cranial walls. I am speaking of such impacts as result not only from the numerous jars, shakings or concussions to which the head is subjected normally by the mere physiologic displacements of its position and by the movements of the body, but also of those more severe impacts to which accident exposes the skull and from which, short of actual surgical trauma, the brain remains uninjured. Further, as a result of the law of physics governing the incompressibility of liquids, the effect of impacts and concussions is diffused over the entire cranial contents, and blows and sudden displacements which might otherwise be followed by serious injury, if limited to a restricted area, have their force thus enormously diminished.

The softness and fragility of the substance of the brain is such that a very special protection against traumatic influences of all kinds, displacements and concussions, is absolutely necessary; hence we have a cavity with hard walls, practically a solid box, in the interior of which the brain is in a condition of hydraulic suspension. The changes which the movements of the head impose on the various surfaces of the brain render a hydraulic suspension especially necessary; the latter, let us bear in mind, exerts its action independently of position.

Liquids transmit pressure equally in all directions; notwithstanding, the gross weight of a column of liquid is in relation, of course, to its height. If the column is placed in a horizontal position there is no inequality of pressure exerted by its weight in any portion of the tube, while in the vertical position the pressure increases as we pass from the upper levels to the base. Changes of pressure must ensue, therefore, in the various levels of the cerebrospinal cavity in variations from the erect to the horizontal position. This fact is in accord with the notable difference observed in the pressure of the cerebrospinal fluid in making lumbar puncture, whether the patient lies down or sits up. It is a legitimate inference, therefore, that the column of cerebrospinal fluid in the spinal canal must in the erect position lessen relatively the pressure within the cranial cavity. As a result of the laws of hydrostatics, the pressure existing in the upper levels, say at

the vertex, must be equal to the pressure existing in the lumbar region less the height of the column which separates the two points. Lessening the pressure in the lumbar region by spinal puncture, must therefore act in a decompressive manner comparable to that of an aspiration, a fact again in keeping within clinical experience. We should bear in mind, however, that in various pathologic conditions, differences of pressure in the levels of the cerebrospinal cavity may be much less than we would be led normally to expect; a fact which, when encountered, as in the instance of a subnormal intraspinal pressure, has in its turn a pathologic significance. The significance of an increased intraspinal pressure will be discussed later. All things considered, however, it should be stated that even under normal conditions the variations in the intraspinal pressure resulting from changes of the position of the cerebrospinal axis from the horizontal to the vertical are much less than we would *à priori* be led to suppose. Possibly, as Bard thinks, this points to some compensatory mechanism for the maintenance of a mean of pressure.

DISTRIBUTION, SOURCE AND EXIT

Leaving for a time the question of pressure, let us consider briefly the distribution of the cerebrospinal fluid, its source or sources, and finally its way or ways of exit from the cerebrospinal cavities. The cerebrospinal fluid is found in the ventricles and in the subarachnoid spaces. The subdural space, if it can be spoken of as such, is bounded by the two apposed moist surfaces of the dura and the arachnoid. It would appear that little if any interchange of fluid takes place between the subdural and the subarachnoid spaces, and further that if there be any passage of fluid it is from the former to the latter. Quincke,[5] for instance, injected cinnabar into the spinal subarachnoid space; none of the cinnabar found its way into the subdural space. However, injected into the cerebral subdural space, it found its way into the subarachnoid space. The pathway apparently was through stomata between the endothelial cells. I believe it probable that under normal conditions no exchange of fluid ever takes place. The arachnoid membrane is distended — ballooned as it were — by the pressure of the subjacent cerebrospinal fluid, and is thus closely apposed to the surface of the dura. Any lesion or break of its continuity would mean a failure of its function.

It is commonly accepted by physiologists and morphologists that the cerebrospinal fluid has its origin mainly in the choroid plexuses of the lateral ventricles, thence it passes through the foramen of Munro

5. Quincke, cited by Weed, T. H.: J. M. Res. **26**:21, 1914-1915.

into the third ventricle, thence through the aqueduct of Sylvius to the fourth ventricle. Here, reenforced by the fluid formed by the choroid plexuses of the fourth ventricle, it passes through the foramina of Luschka and the doubtful foramen of Magendie to the subcerebellar cisterna. One of the surprising facts in this connection is the small size of the aqueduct of Sylvius. According to Weed, too, the amount of fluid obtained by catheterizing the aqueduct is very small. Clinically and pathologically, of course, we know that obstruction of the aqueduct leads to dilatation of the lateral and third ventricles, a fact which has also been confirmed by the experimental obstruction of the aqueduct in dogs by Frazier and Peet.[6] The inference from the facts would seem to be that the flow of fluid through the aqueduct, while it undoubtedly takes place, is very slow. Similarly, we are impressed by the exceedingly small size both of the foramina of Luschka and of the foramen of Magendie; indeed, as regards the latter, anatomists are not wanting who regard it as an artefact and as having no real existence; and as regards the foramina of Luschka, Lochelongue[7] states that they are not absolutely constant. Is there any communication through the transverse fissure, as is claimed by Lochelongue? However this may be, we are forced to conclude that, in any event, such passage of fluid as takes place into the basillar cisternae must be quite slow. Frazier and Peet,[6] for instance, measuring the escape of the fluid in the dog by placing a cannula through a small incision in the occipito-atlantoid ligament, determined that in one instance the amount averaged about 0.231 c.c. per minute, and in another 0.192 c.c. per minute, certainly a very small amount.

In addition to such fluid as escapes into the subcerebellar cisterna, some fluid doubtless passes into the central canal of the spinal cord; here again we are impressed by the minute size of the canal and the fact of its not infrequent obliteration. Finally, the cerebrospinal fluid probably passes freely into the spinal subarachnoid space.

SOURCES OTHER THAN THE CHOROID PLEXUSES

The question arises, has the cerebrospinal fluid any other origin than the choroid plexuses? Most physiologists reply that very little fluid can be ascribed to other sources. Frazier and Peet believe that it is generated only within the ventricles. Some writers, however, attribute some of the fluid, though a small portion, to drainage from the perivascular and perineuronal spaces. I myself, however, believe — and I am not alone in this — that the assumption of a communica-

6. Frazier, C. H., and Peet, M. M.: Am. J. Physiol. **35**:268, 1914.

7. Lochelongue: Le Lequide Cephalo-Rachidien et Ses Anomalies, Paris, A. Maloine et Fils, Editeurs, 1918, p. 8.

tion, at least a direct commuuncation, between these spaces and the subarachnoid space is not justified. Spina[8] induced an artificial hyperemia in the brains of dogs by ligating the aorta high up and giving injections of suprarenal extract. He observed a transudation of clear pearly drops of liquid on the brain surface. He regarded this liquid as cerebrospinal fluid. Frazier[9] also observed drops of cerebrospinal fluid appear on the surface of the brain under high arterial tension. While these experiments and observations may and probably do point to a source of the cerebrospinal fluid other than the choroid plexuses, they do not justify the inference that a communication exists between the perivascular spaces and the subarachnoid space as is inferred by Weed. Weed[5] succeeded in injecting the perivascular spaces with prussian blue from the spinal subarachnoid space after exsanguinating the animal. This procedure, however, is open to the objection that in an intense anemia as in an intense congestion produced by artificial means, various structures may be broken down. In other tissues far more robust and resistant than brain tissue, even capillary hemorrhages may occur under such conditions. Further, Weed's experiment consisted in a veritable "aspiration" of the nerve tissue; indeed, he himself uses the term "ferrocyanid aspiration." Here, again, the objection holds good that structures may be broken down by violence to the normal relations of pressure. This becomes especially probable when we consider the extremely delicate character of the structures involved. For instance, Weed speaks of the perivascular spaces as tubes "lined by mesothelium, surrounding the arteries and veins as far as the finer subdivisions into capillaries. Whether lined by mesothelium or simple glia cells is still in doubt." And, again he says, "Both the perineuronal and capillary space are unlined except by the loose stroma of the nervous tissue." Surely, we have here structures exceedingly vulnerable to traumatic influences, no matter how slight. Further, it becomes evident that in Weed's experiments the prussian blue granules also found their way into structures other than the perivascular and perineuronal spaces; for Weed tells us that "No prussian blue granules can be made out in any definite relation to the individual neuroglia cells, but the generalized distribution while small in amount, as compared to the collection in the perivascular system, undoubtedly indicates that this system is connected also with the supporting neuroglial fibrillar structures of the nervous tissue." In other words, Weed was obliged to enlarge his conception of the perivascular spaces so as to include the neuroglia as well. Does it not seem more probable that the

8. Spina: Wien. med. Bl. **21**:247, 265, 1898; Arch. f. d. ges. Physiol. **76**:204, 208, 213, 1899; ibid. **80**:370, 1900.

9. Frazier: Surgery of the Spine and Spinal Cord, New York, D. Appleton & Co., 1918, p. 143.

prussian blue granules were forced into places between which and the subarachnoid space no normal communication exists? And if this be true of the neuroglia may it not be true of the perivascular, the pericapillary and the perineuronal spaces as well? Finally, even admitting the validity of both Spina's and Weed's experiments, they would merely indicate the passage of material under certain conditions from the subarachnoid space into the perivascular spaces, and not from the perivascular spaces into the subarachnoid space; and it may be safely maintained that no evidence exists to prove the passage of material in the latter direction. We have just seen that while communications exist between the subdural space and the subarachnoid space, this communication is in only *one* direction, namely, from the subdural space into the subarachnoid, but not in the reverse direction. Surely in the present instance it is unjustifiable to assume, because under abnormal conditions pigment granules can be forced into the perivascular and perineuronal spaces and also into the neuroglia, that therefore a passageway normally exists and a flow normally takes place from these spaces into the subarachnoid spaces. To the consideration of this subject I will return a little later.

The principal source of the cerebrospinal fluid is, as we have seen, ascribed by physiologists to the choroid plexuses. If, as I believe probable, drainage material from the perivascular spaces is to be excluded, the question arises as to whether there are any other sources of the fluid. Obviously, the membranous surfaces suggest a source of supply. Physiologists in general are inclined to ascribe to these surfaces a very insignificant rôle. However, analogy would suggest a degree of such function on their part. We have merely to call to mind the synoviae of the joints, the peritoneum, the pleura, the pericardium and other serous surfaces. In the instance of the subdural space, the facts justify the inference that the fluid present, though small in amount, is not derived from the subarachnoid space. We again call to mind the exceedingly small avenues of communication between the ventricles and the subarachnoid spaces of the brain and spinal cord. It certainly seems strange that the large amount of fluid in the various cisternae and especially in the spinal canal should all find its way through the foramina of Luschka and the apochryphal foramen of Magendie. Further, a fact to which Cushing also calls attention[10] is that the ventricular and spinal fluids are not exactly identical in composition; for the ventricular fluid contains a higher percentage of sugar than the spinal fluid, while the spinal fluid contains a higher percentage of globulin. It is difficult to account for these differences by a single source of supply. Is it not possible that

10. Cushing, H.: J. M. Res. **26**:1, 1914-1915.

the rôle which the membranes play is greater than that commonly supposed? When we call to mind the occasional behavior of other serous surfaces — it is true under pathologic conditions — the idea does not seem so very improbable; and while the arachnoid itself has no vascular supply, its intimate neighbor, the pia, is richly endowed with vessels. Spina's experiments on the results of artificially induced cerebral hyperemia may have here a special significance.

The next question that presents itself is as to the way in which the cerebrospinal fluid makes its exit from the cerebrospinal cavity. The admirable researches of Weed have shown that the cerebrospinal fluid makes its exit, in very large part, by means of the arachnoidal villi into the sinuses, i. e., into the venous system of the dura. The arachnoidal villi are extremely delicate structures which project into or are in close relation to the venous lacunae or with the lumen of the sinuses. As to the method of transition, Weed states that it becomes evident in the study of the mesodermal cells, which exist between the myxomatous groundwork of the villus and the lumen of the sinus, that the escape of fluid from the villi is probably through the cell substance. Weed uses the term "filtration" in describing the process; while this term appeals to me because it implies a purely physical process, it is perhaps objectionable on the ground that a filter removes or holds back something from a liquid undergoing filtration, and there is nothing in the functioning of the arachnoidal villi to indicate such a process. Perhaps the word osmosis or diffusion, both of which Weed mentions, would be more applicable; or better still, perhaps the word absorption would meet the facts best, as it is really a process of absorption of the cerebrospinal fluid by the venous blood. Weed also concludes, and in this he is in accord with other investigators, that in addition to the major return of cerebrospinal fluid by the arachnoidal villi there is also an accessory drainage of fluid into the lymphatic system, though he believes that this plays a comparatively insignificant part in absorption except from the isolated spinal subarachnoid space; further, he concludes that absorption from the cranial subarachnoid space is much more rapid and much greater in amount than from the spinal portion. He also concludes, and this is most important, that no evidence has been afforded, in his observations, of the escape of cerebrospinal fluid into the cerebral veins or capillaries.

The lymph channels which aid in the escape of the cerebrospinal fluid are those in the sheaths of the cranial and spinal nerves. As just stated, these channels are mainly active in the spinal subarachnoid space, though there is no good reason for denying them this rôle in the cranial subarachnoid space. Perhaps in the spinal region they are the sole means of exit. According to Halliburton, diffusion of the

cerebrospinal fluid is most rapid in the subcerebellar district and extremely slow in the spinal, especially in the lower spinal region. Halliburton also states, and in this he is likewise in accord with others, that diffusible substances introduced into the subarachnoid space pass rapidly into the veins.[11]

The last mentioned fact is of enormous practical value, for it shows that attempts to medicate the central nervous system, as is done especially in the intraspinal injections of arsphenamized serum in the Swift-Ellis method, are essentially unscientific and unphilosophical. The exceedingly minute — indeed, apochryphal — amount of arsphenamin contained in the serum is speedily removed by the dural sinuses and the lymphatic channels of the nerves, and never enters the nerve tissue. Still further diluted and weakened by the spinal fluid, it can at most pass over the membranes in its journey of exit. That the benefit which is supposed to result from the method is really due to another and entirely incidental factor will, I believe, become apparent as we proceed.

RELATIONS TO THE VASCULAR AND LYMPHATIC SYSTEMS: QUESTION OF NUTRITION OF NERVE TISSUE

Let us now turn our attention once more to the relation of the cerebrospinal fluid to the vascular system and also to the lymphatic system. We have already learned that the cerebrospinal fluid is in direct relation with the venous system through the arachnoidal villi and the dural sinuses; also, that it is in direct relation with the lymphatic system of the cranial and spinal nerves. What is its relation to the perivascular and perineuronal spaces? I have already advanced reasons for denying the presence of the communication assumed to exist between these spaces and the subarachnoid space, and denying them the rôle of being merely one of the cerebrospinal fluid containers. Let us for a moment call to mind some of the elementary facts of structure and physiology. Everywhere, in all of the organs and tissues, whether we think of the Haversian canals of the bones, or of the lymph sheaths which surround the blood vessels in the muscles, or of any of the other structures, we find that the nutrition of the tissues takes place not directly from the blood vessels, the capillaries, but through the intermediation of lymph channels. As Halliburton[11] expresses it:

> The lymph acts as the intermediary or middleman between the blood and the tissue elements, conveying to the latter on the one hand the oxygen and the nutritive substances they need; and on the other, it is into the lymph primarily that the tissues pour the waste products of their activity.

11. Halliburton, W. D.: The Possible Function of the Cerebrospinal Fluid, Brain **39**:214 (Oct.) 1916.

Why should we deny this rôle to the perivascular, pericapillary and perineuronal spaces? Weed sees an objection to considering these spaces as lymph spaces, saying:

> If this were the case, we should expect a content in lymph — cellular and containing many coagulable elements. The protein of such a tissue juice of lymph should suffice to keep patent the perivascular spaces after fixation for microscopic work. Ordinarily, however, these spaces are not distended, indicating clearly that the coagulable elements are very slight.

I would answer that these appearances are exactly those that are encountered in conditions in which the metabolism of the nervous tissue is increased, as occurs in certain pathologic conditions; notably in paresis in which even in relatively early stages the perivascular spaces are found enlarged and containing many formed elements. Further, the changes that take place normally during the process of nutrition in the lymph spaces surrounding the blood vessels of the various tissues are not such as are revealed by the microscope. The tissues receive their nourishment not from proteins in the lymph spaces but from amino acids which are the products of the cleavage of the proteins circulating in the blood vessels. In fact, this is true of all of the substances concerned in nutrition unless it be the mineral salts. The fats are split into alcohol and fatty acids, complex carbohydrates are fragmented, albumin is split up into peptones, the latter are reduced to amino acids and the amino acids into still simpler components. I submit, again, that such changes are not revealed by the microscope. Further, to include the perivascular spaces in the general cerebrospinal fluid space, of necessity, makes the content of the perivascular spaces the same as the content of the subarachnoid space and the content of the ventricles; in other words, it fills the perivascular spaces with cerebrospinal fluid. However, complex chemical changes, such as are concerned in nutrition, do not and cannot go on in the cerebrospinal fluid. The cerebrospinal fluid contains no ferments and can develop no ferments. It will not mend matters to say that the physicochemical changes of nutrition take place in the blood plasma and within the blood vessel walls, for the cells of the various tissues are the active agents which reduce — that is, split up — the materials offered by the blood plasma into components adapted to and peculiar to their individual structure; but between the cell and blood plasma lies the lymph space. Obviously, the change must go on through the lymph space; it cannot be otherwise. Surely, if the cerebrospinal fluid is the nutrient fluid of the brain, it should bear in its constitution some evidence of this fact.

Speaking of the perivascular spaces, Weed thus expresses himself:

This whole accessory fluid system of the cerebrospinal canal axis—an intramedullary canalicular system—undoubtedly possesses an active function in maintaining the metabolic exchange and elimination of the nerve-cells. Throughout the body in the other tissues there is a chief and accessory circulation. For in these other tissues of the body there is in addition to the blood capillary a lymph capillary with the tissue juice or plasma playing the intermediate part in exchange.

Notwithstanding, in keeping with the long prevalent — and to my mind, erroneous—belief, he declares that "nervous tissue lacks entirely the lymphatic system, and that it would appear that its place is taken by the perineuronal, pericapillary and perivascular system." This, it seems to me, is a distinction without a difference, and it clearly has its origin in the mistaken view that the perivascular spaces share with the subarachnoid spaces and the ventricles the function of being cerebrospinal fluid containers. It is interesting to note in this connection that Mott,[12] in his Oliver-Sharpey Lectures on the Cerebrospinal Fluid, treats the perivascular space as a lymph space, though he believes that it communicates with the subarachnoid space. Speaking of the perivascular space, he says:

It is filled by a *clear and transparent fluid* which from the point of view of its morphological significance should be considered as lymph. It contains in variable amount lymph corpuscles, fatty granules and sometimes even drops of oil.

Mestrezat[13] likewise maintains that the perivascular spaces constitute a lymphatic circulation, and, further, that this lymphatic circulation consists of a true lymph distinct from the cerebrospinal fluid and that no mixture of the two fluids takes places.

Unfortunately, the erroneous view that the perivascular spaces are filled with cerebrospinal fluid has given rise to the entirely untenable position that the cerebrospinal fluid is the nutritive fluid of the central nervous system. This is the view held, for instance, by both Weed and Halliburton. Halliburton tries to overcome one of the obvious difficulties by declaring that the amount of protein necessary for the repair of the nervous tissues is not great, implying that the amount of metabolism in nervous tissue is exceedingly small. However, it is probably much larger than Halliburton would lead one to suppose. What is the meaning of the enormously rich vascular supply to the brain and cord, if it does not predicate a large amount of nutritive change? What is the meaning of the enormous mass of capillaries if

12. Mott, F. W.: The Oliver-Sharpey Lectures on the Cerebrospinal Fluid, Lancet **2**:1, 79, 1910.

13. Footnote 3, p. 188.

it does not imply an active metabolism? Indeed, one might ask, what is the need of a vascular supply at all if the nutrition of the central nervous system is carried on by the cerebrospinal fluid?

It cannot be but that the nutrition of the nervous system is in keeping with that which goes on elsewhere in the body, and that definite chemical and physical changes take place in it as in the other tissues. In muscle, for instance, the chemical reaction, which is neutral or feebly alkaline when the muscle is at rest, becomes acid in a muscle that is active; there is also in the active state a greatly increased elimination of carbon dioxid while the tissue consumes proportionately more oxygen; further, the active muscle contains more water, it yields an augmented quantity of extractives soluble in alcohol, a lessened quantity of extractives soluble in water, a lessened quantity of substances producing carbon dioxid, a lessened quantity of fatty acids and of creatin and creatinin, and a lessened quantity of glycogen. Its circulatory relations are modified; its capillaries dilate; it contains more blood, and the blood in its veins differs chemically from that found in its veins when at rest.

Nerve substance, like muscle, when at rest is neutral or feebly alkaline, and when active is acid. It has not, however, been possible to determine the chemical changes in detail. Not even the exchange of oxygen and carbon dioxid has been demonstrated, though the facts attending vascular supply and temporary obstruction justify the inference that such changes of necessity take place. The effect of temporary compression of the vessels of the brain or of the abdominal aorta in producing arrest of function — of the brain, on the one hand, or of the cord, on the other — is an instance in point. Halliburton[14] himself admits that the nerve centers are richly supplied with blood vessels which furnish them with an abundant supply of nutrient material, that cerebral anemia rapidly produces pathologic changes in the nerve cells, and that death quickly supervenes. Further, a number of observers have demonstrated that nerve cells at rest and in activity present marked differences histologically. Thus Hodge, Vas, Nissl, Mann, Lugaro and others have studied the changes in nerve cells that result from function. While the various investigators differ somewhat as to details, they agree as to the essential facts. They have discovered that during rest the chromatic substance in the cells increases in amount, and that during functional activity it diminishes. Activity of the nerve cell is first accompanied by swelling of the protoplasm of the cell body, later by a progressive diminution in the size of the cell body. If functional activity be prolonged, the nucleus undergoes changes

14. Halliburton, W. D.: Lectures on Biochemistry of Muscle and Nerve, Philadelphia, P. Blakiston's Son & Co., 1904, p. 79.

similar to those of the cell body, and this is true also of the nucleoli; there is at first an increase in volume, which subsequently gives way to the reducing action of fatigue. Halliburton[14] tells us that cholin, derived from the decomposition of lecithin and other phosphorized fats, is obtained from perfectly fresh nervous tissues, that its presence is a very positive sign of chemical activity and that the gray matter yields the most cholin. Unfortunately, little is known of the chemical changes in the blood of the venous return; but certainly such facts as are in our possession are in favor of metabolic changes taking place in nerve tissue of no mean degree of activity. This is also in keeping with the enormous activity of the nervous centers which, with the possible exception of those portions concerned in consciousness, is incessant. To me the conclusion is more than justified that the chemical changes taking place in the central nervous system are, to say the least, considerable, and that they take place not through the cerebrospinal fluid but through the blood vessels and in the spaces which surround the latter, and that the function of these spaces is indistinguishable from the perivascular lymph spaces existing elsewhere. These considerations are, as we will see, not of theoretical interest purely, but of great practical importance. Finally, a conclusive proof that there is no communication between the perivascular, pericapillary and perineuronal spaces on the one hand, and the cavities that contain the cerebrospinal fluid on the other, is furnished by the fact that the cerebrospinal fluid normally contains no products of nerve tissue metabolism. Halliburton[15] tells us that normally it contains no cholin, no cholesterol, no nucleoprotein.

PRESSURE RELATIONS: SPINAL DRAINAGE

Let us now turn our attention to the pressure under which the cerebrospinal fluid exists; the relations of this pressure to that of the arteries, capillaries and veins, and to that in the dural sinuses; also to the movements of the cerebrospinal fluid, and, finally, to questions of its renewal, all of which questions have a practical bearing.

We have already considered in part the question of the pressure of the cerebrospinal fluid in relation to its hydrostatic properties. Studies and estimates of the pressure of the cerebrospinal fluid have been made by a large number of observers. The figures that are given disclose a wide range of variation; for example, Cybierski would place the pressure at from 72 to 90 mm. of water, while Bard would place it at 200 mm. Perhaps it is fair to conclude, as does Richet[16] from averaging the results of Quincke, Krönig, Parizot and Boveri, that it is

15. Halliburton: Handbook of Physiology, 1919, p. 168.
16. Footnote 7, p. 53.

about 100 mm. Doubtless the differences in figures are due in large part to differences in the methods of investigation employed. For us, however, the fact of physiologic importance is that the cerebrospinal fluid normally exerts a pressure on the walls of its containers, and that this pressure is greater than that in the venous sinuses; absorption by the latter of the cerebrospinal fluid through the arachnoidal villi is thus directly favored. The pressure of the cerebrospinal fluid is also greater than that in the lymphatics,[17] which fact likewise favors the theory of absorption. It should be added that Bard estimates the pressure in the cerebral capillaries to be six or seven times as great as that of the cerebrospinal fluid; so that it is extremely improbable that any absorption takes place into the capillaries as is supposed by Mott. The pressure in the cerebral arteries is also notably higher than that of the fluid; that in the cerebral veins is clearly higher than that in the sinuses. The cerebral arteries, capillaries and veins, it should be borne in mind, have no relation of absorption with the cerebrospinal fluid. The relations of the latter are merely with the sinuses and with the lymphatics of the cranial and spinal nerves.

The pressure of the spinal fluid is doubtless maintained by a continuous supply. The latter is furnished as we have seen by the choroid plexuses[18] though other sources are not excluded. The questions arise,

17. Footnote 7, p. 54.

18. Bard (Footnote 4), after commenting on the numerous plications of the choroid plexuses, estimates the surface of the latter as equivalent to 1 square meter.

Bard thinks that the vascular richness of the plexuses permit of great variations in volume and that this exerts beyond a doubt an influence on the pressure of the liquid in which the plexuses are immersed. He is led to think, further, that the regulation of the spinal fluid pressure may depend on two different mechanisms, one for changes slow and persistent, and another for changes sudden and rapid, such for example as are caused by changes in attitude. The first depends on the quantity of cerebrospinal fluid present, as determined by the relative rates of formation and absorption. The second, Bard thinks, is a function of the pineal gland which he has come to regard as a barometric perceptor and regular. Its action would seem to influence the distention and activity of the choroid plexuses. The discussion of this interesting subject would, however, take us too far afield.

Bard also points out that the pressure of the cerebrospinal fluid is in relation with the external atmospheric pressure through the round window of the internal ear, and suggests that perhaps changes in the pressure of the fluid could be clinically studied in the ear, thus rendering a spinal puncture unnecessary. Personally it is not clear to me how the round window could be observed. However, the pressure of the fluid is also in relation with the oval window. Is it possible, perhaps, that transmitted through the chain of bones, changes of pressure could be studied through the drum membrane?

What is the total amount of cerebrospinal fluid and how often is it renewed? The total amount of cerebrospinal fluid has been estimated at from 125 to 150 c.c. It appears to be undergoing a gradual and constant renewal. Mestrezat, Frazier and others believe that it renews itself six or seven times in twenty-four hours. I am inclined to think that this estimate is excessive, and that normally the spinal fluid renews itself much more slowly. The flow of cerebrospinal fluid observed every now and then in fractures of the cranium, in disease of the ethmoid, and in other pathologic states in which the subarachnoid space is in communication with the external world — a flow which is sometimes extraordinary in amount — must not be taken as the measure of the fluid normally produced, but should be looked on as an effort on the part of nature to maintain the normal degree of pressure by an excess of secretion, i. e., an effort to overcome the leakage. This fall of pressure caused by the leakage results, it would seem, automatically in an increased secretion. This fact — namely, of a fistulous opening greatly stimulating the formation of cerebrospinal fluid — has an important bearing on the question of a therapeutic expedient which has for a number of years past been practiced extensively in my clinic at the Jefferson Hospital, namely, spinal drainage. The flow which follows a lumbar puncture is at first very free; very soon the flow moderates and the fluid escapes from the cannula by a series of drops. Gradually these become less frequent until finally the fluid escapes in drops formed at the end of the cannula at intervals of several seconds, five or longer. It is interesting to observe that the final rate no longer changes, but is steadily maintained, no matter how long the cannula remains in position. In other words, the puncture has not only permitted the escape of fluid in an amount equivalent to a fall of pressure equal to that of the atmosphere, but has also, and by virtue of this very fact, stimulated the production of a fresh amount of fluid. In this way spinal drainage results, when practiced thoroughly, in a veritable washing out, a lavage, of the subarachnoid space.

MOVEMENTS: MAINTENANCE OF PRESSURE: NATURE OF THE FLUID

Before considering spinal drainage in detail and in order to complete our study of the functions of the cerebrospinal fluid, let us briefly consider its movements, the causes of the maintenance of its pressure, and, finally, its nature. It is exceedingly probable, as already pointed out (page 234), that the cerebrospinal fluid passes from the cranial subarachnoid space into the spinal subarachnoid space. Does it remain there to be disposed of by gradual absorption by the lymphatics of the spinal nerves, or is there a return flow into the cranial cavity, and does the one time spinal fluid share in the exit of the cranial fluid

through the arachnoidal villi? If there is a return flow, what is the pathway? These questions cannot be definitely answered. The difference between the chemical constitution of the cerebral and spinal fluids, which has already been commented on, though slight, is opposed to a free commingling of the fluids such as would be suggested by an actual return flow. It is true, as has already been stated, that, under experimental conditions in animals, pigment granules injected into the spinal subarachnoid space find their way into the cranial cavity, but the condition of the experiments, as has been pointed out, are such as to lend no justification to the inference that such a passage actually occurs normally.

We are all familiar with the phenomenon of brain pulsation, synchronous with the heart's action, a pulsation visible to the eye on the exposure of the brain at the time of operations, and also palpable in the fontanels of the infant and in adults in whom a portion of the cranium has been lost. What happens in the closed cranium at the time of a systole? Both the cerebrospinal fluid and the blood being liquids, they must obey the physical law of incompressibility. Something, however, must yield. What is it? Bard believes that at the moment of systolic arrival of the arterial blood, a pulse wave is created in the cerebrospinal fluid as a result of which cerebrospinal fluid flows — is driven — into the vertebral canal, that this outflow compensates for the inflow of blood, and that this is followed during diastole by a flow of the fluid in the reverse direction. He states that the outflow of the fluid has been demonstrated by François-Franck by placing a hemodrometric pallet in the occipito-atloidian space. Cathelin disputes the reality of this oscillation, and claims that the outflow of fluid alone takes place and that the fluid leaving the cranial cavity continues its exodus without turning backward. It is a little difficult to accept the theory that at the moment of systole, cerebrospinal fluid is driven from the cranial cavity into the spinal cavity. In the first place, the latter is already full and its walls can hardly be said to be more distensible than those of the cranial cavity, and, further, its arteries are also conveying blood into the spinal cavity at the same moment that blood is entering the cranial cavity. Is it not more likely that of the contents of the cranium, it is the tissue of the soft nonresisting nervous system which in some way adapts itself to the increased pressure of the arterial wave? This I believe is the only possible explanation. Brain tissue as a whole is readily compressible, and probably the increase of pressure caused by entrance of blood at the moment of systole is accompanied by a yielding — not on the part of the neurons and nerve fibers — but by a yielding on the part of the perivascular spaces and perhaps in a degree by a yielding of the soft neurogliar tissue. Two things would result from such a mechanism: first, the blood, itself

incompressible, would be driven into the soft tissue of the brain, the latter immersed in an incompressible medium, the cerebrospinal fluid, with relatively great force. In other words, the mechanism by means of which the brain tissue is placed between two incompressible mediums would result in the capillaries being very thoroughly filled at each systolic wave. Second, it is exceedingly probable that the contents of the perivascular spaces are gently moved onward at the same time; besides, the perivascular spaces afford the space necessary for the play of the expanding and contracting blood vessels. That the perivascular spaces probably terminate, as do the perivascular lymph spaces of all other tissues, in channels in or about the walls of the blood vessels and perhaps in other ways not yet recognized, and so make their exit from the cranium, is to my mind exceedingly probable.[19]

Respiratory movements, as is well known, also influence the movements of the intracranial contents, a fact which is familiar to every one who has practiced lumbar puncture. The respiratory pulse wave, however, makes itself evident only through the intermediate action of the vascular apparatus and the problems presented in no wise differ from those just considered and need not detain us.

What are the causes of the maintenance of pressure of the cerebrospinal fluid? Evidently these must be sought in the continuous formation of the fluid. I have already commented on the rôle played by the choroid plexuses. There has been a tendency of late to speak of the choroid plexuses as the choroid "glands," and of the cerebrospinal fluid as though it were a glandular secretion. But is it proper to speak of it in this sense? A glandular secretion is a fluid containing new bodies, new chemical principles, ferments, hormones or other substances not previously existing in the blood, but which have been elaborated from the blood plasma by the specific action of glandular cells. Assuredly, the cerebrospinal fluid presents no such constitution. Indeed, as we have seen, it presents little more than the three quarter per cent. common salt solution of the histologic laboratory. Mestrezat, because of the inorganic constitution of the cerebrospinal fluid, terms it a "mineral serum" and contends that its constituents exist preformed in the blood plasma and that they are derived from the latter by a

19. Admitting for the sake of argument that the perivascular spaces are really cerebrospinal fluid containers, the question arises, Do these spaces leave the blood vessels at some hypothetical point or points to enter, like so many tubes, the subarachnoid spaces? and if so, Are the blood vessels distal to such point or points, either in the nerve tissue or in the pia, devoid of spaces—lymph or other—about or in their walls? Such a supposition is obviously absurd and we have here another reason for regarding the perivascular spaces as being related intrinsically and essentially to the blood vessels.

process of dialysis pure and simple through the epithelium of the choroid plexuses. Lochelongue,[7] it should be added, also holds to the view that the cerebrospinal fluid is not a true secretion.

ACTION OF THE CHOROID PLEXUSES

It would appear that the action of the choroid plexuses, and more particularly of their investing epithelium, is to permit the passage into the cerebrospinal fluid of water and of such a minute quantity of salines and other substances as are necessary to preserve the innocuous and neutral character of the fluid, and also its specific gravity, properties so necessary to its service as the medium of hydraulic suspension of the central nervous system. The passage of all other substances is inhibited or prevented. The inability of various substances thrown into the circulation to find entrance into the cerebrospinal fluid, a fact demonstrated by Frazier[9] and others, is well known. This fact, however, does not mean that it is the function of the choroidal epithelium and of the lining mesothelium of the subarachnoid spaces to prevent toxic substances from reaching the central nervous system, but merely to *prevent such a change in the composition of the fluid as would interfere with its action as the innocuous medium of hydraulic suspension*. Bard characterizes the choroidal epithelium as a simple barrier which prevents little else than water and sodium chlorid from passing through.

UNSCIENTIFIC CHARACTER OF INTRASPINAL MEDICATION

The mistaken view that the nutrition of the nervous system is carried on by means of the cerebrospinal fluid has led to attempts to utilize the cerebrospinal fluid of the spinal subarachnoid space as a medium of medication. Noticeably has this been the case in the treatment of paresis and tabes by intraspinal injections of so-called arsphenamized serum, the Swift-Ellis method. The belief has been that by this means the obstruction which the choroid plexus is supposed to offer to the arsphenamin could be circumvented. I have already pointed out that substances introduced into the cerebrospinal fluid rapidly disappear, passing through the arachnoidal villi into the venous system without entering the parenchyma of the central nervous system in the slightest degree. If arsphenamin acts at all when introduced in simple solution into the spinal subarachnoid space, it is by irritating and inflaming the spinal membranes, and, in a variable degree, the other spinal contents. This is the legitimate inference to be drawn from the experience of Paul Ravaut[20] and of others; unless at least

20. Ravaut, P.: Ann. de méd. **1**:49, 1914.

the arsphenamin has been used in such small amounts as to be under any circumstances practically valueless. These conclusions doubtless apply with equal force to the introduction of corrosive sublimate and other antisyphilitic remedies via the subarachnoid route.

I am, of course, well aware of the claims made in behalf of the Swift-Ellis and allied methods, especially in the treatment of tabes, of spinal syphilis of the exudative form, and to a less extent of paresis. For a long time I made use of this method most extensively in my clinic and in my private work, and I can confirm the claims made as to undoubted improvement in tabes and spinal syphilis, and of apparent improvement from time to time in paresis. I have, however, for several years past come to the conclusion that the improvement does not depend on the intraspinal injection of the arsphenamized serum, but on the drainage of the spinal subarachnoid space which must inevitably be practiced before the injection is made.

SPINAL DRAINAGE: ITS MODE OF ACTION

Several years ago, Dr. Sherman F. Gilpin, chief of my clinic, conceived the idea that it would be a good plan to practice spinal drainage at the time of giving arsphenamin intravenously in the hope that, the intraspinal pressure being reduced, the arsphenamin circulating in the blood would be more likely to diffuse into the subarachnoid space. Later, Dr. Gilpin together with Dr. Thomas B. Earley instituted simple spinal drainage alone. The results were striking. Marked improvement followed, an improvement which was more pronounced than that observed in the Swift-Ellis method, doubtless because the drainage was more thorough. The improvement was most decided, as might have been expected, in tabes, but was also very evident in paresis. The patients are put to bed and drained of every possible drop of fluid about once in a week, once in ten days, or once in two weeks. The mode of operation of the procedure is probably as follows: To begin with, there is an immediate reduction of the increased intradural pressure; secondly, the drainage results in a kind of lavage of the dural space, for the spinal fluid which has been removed is rapidly replaced. In keeping with this fact, there is a fall in the lymphocyte count. Further, there is normally a balance between the pressure in the blood vessels within the cord and the pressure of the surrounding cerebrospinal fluid. It follows that if the pressure of the cerebrospinal fluid is abnormally increased, the vascularity of the cord will be correspondingly diminished; less blood than normally will be able to enter its vessels. It would appear, therefore, that the rapid withdrawal of the cerebrospinal fluid is followed by an increase in the vascularity of the cord — a relative hyperemia — and other things being equal, of the

brain as well. Probably the results — often truly remarkable — which follow radical spinal drainage are to be attributed to the improved nutrition following this increased vascularity. We have here a parallel to the results of the Bier method in surgery. It should be added that we have never noted any untoward results from spinal drainage in either paresis or tabes. That the diagnosis — previous to drainage — should definitely exclude brain tumor and brain abscess goes, of course, without saying. Finally, it is interesting to note that the reason the pressure of the cerebrospinal fluid is pathologically increased in tabes and paresis is that its outflow is obstructed. It would appear that the inflammation of the membranes, the meningitis, present in these affections leads to an occlusion more or less pronounced of both the arachnoidal villi and the lymphatics. The cerebrospinal fluid continuing to be formed, a rise of pressure necessarily follows. Spinal drainage is, therefore, directly indicated in both tabes and paresis.

For a long time past, we have abandoned the Swift-Ellis method in my clinic. We have relied altogether on intravenous injections of arsphenamin and mercurial inunctions together with spinal drainage, and our results have been better than previously, doubtless because we have given especial attention to the drainage, removing at each occasion all the spinal fluid possible. Finally, in a number of cases, we have practiced spinal drainage alone, and always with improvement.

It is on the incidental spinal drainage, practiced in the Swift-Ellis and allied methods, let me repeat, that such success as has attended the procedure depends, and not on the apochryphal amount of arsphenamin contained in the serum. I am entirely in accord with the position assumed by B. Sachs[21] as to the uselessness and unnecessary character of the method. Besides Sachs tells us that with Professor Benedict he has succeeded in showing that arsphenamin introduced in the usual quantities into the blood current appears in the cerebrospinal fluid in appreciable quantities. Sicard and Bloch[22] have likewise noted this fact, the arsenic making its appearance in the cerebrospinal fluid an hour after the intravenous injection. Kopke[22] also noted the presence of arsenic in the cerebrospinal fluid after the administration of atoxyl in trypanosomiasis. Even admitting, therefore, that the presence of arsphenamin in the cerebrospinal fluid is of any consequence, no pretext can any longer exist for the intraspinal injections of arsphenamized serum.

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21. Sachs, B.: On the Intravenous, Not the Intraspinal, Use of Arsphenamin in Syphilis of the Nervous System, Arch. Neurol. and Psychiat. **1:277** (March) 1919.

22. Cited by Mestrezat (Footnote 3) p. 93.

THE REAL DIFFICULTIES OF ANTISYPHILITIC MEDICATION

The difficulty with the various antisyphilitic remedies at our command is not that when introduced by the mouth or parenterally, they do not pass through the epithelium of the choroid plexuses, but because they do not pass through the walls of the capillaries. The reason that the iodids, mercurials and arsphenamin are so efficacious in syphilis of the exudative, the gummatous, form, is because in this form the spirochete has invaded especially the lining membrane, the walls of the blood vessels and the immediately contiguous tissues. These structures are all of them directly accessible to the action of remedies circulating in the blood. The walls of the capillaries, however, offer an obstruction to their entrance into the pericapillary and perineuronal spaces. Therefore, no action by such remedies can take place on the nervous parenchyma. How seriously the latter is invaded in both paresis and tabes, we all know. It is here that the difficulty lies and not in the barrier offered by the choroidal epithelium. An element of hope is presented in that the walls of the blood vessels do not offer the same rigid barrier as does the choroidal epithelium so necessary to the preservation of the integrity of the cerebrospinal fluid. We know that the capillaries do permit various drugs to pass through; among these are the alkaloids; morphin; cocain; caffein; thein; quinin; various salts, such as the bromids; various narcotics, such as trional, sulphonal; various urea derivatives, such as veronal, medinal, luminal, besides urotropin and the volatile anesthetics; doubtless many other substances could be enumerated.[23] It would seem that the walls of the capillaries are especially impervious to substances containing the ions of the metals. Two things appear to be necessary to the conquest of parenchymatous nervous syphilis: one, an increased knowledge of the biology of the *Spirochaeta pallida*, and the other, the development of a diffusible and innocuous spirochetocide based on this knowledge. In other words, the problem is one of biology and biochemistry.

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23. Halliburton writes, "If, as Weed has suggested, the cerebrospinal fluid is partly formed, as ordinary lymph is, by exudation from the blood into the perivascular spaces, it is difficult to understand why readily diffusible drugs and poisons do not escape readily into the cerebrospinal fluid as they do into ordinary lymph," Brain, loc. cit., p. 219. As the writer points out above, many drugs and poisons undeniably diffuse through the capillaries and the pericapillary and perineuronal spaces, and the reason they are not found in the cerebrospinal fluid is that these spaces do not—as is commonly supposed—drain into the cerebrospinal fluid. All difficulties disappear when this mistaken interpretation is abandoned.

CONCLUSIONS

Leaving this digression, let us briefly summarize the conclusions which the foregoing considerations of the cerebrospinal fluid appear to justify.

First, the cerebrospinal fluid is preeminently a fluid for the hydraulic suspension of the brain and cord; its function is essentially hydrostatic.

Second, its chemical constitution is essentially that of the innocuous three quarter per cent. common salt solution of the histologic laboratory. It has no action on the tissues with which it comes in contact: it is absolutely neutral and negative.

Third, it is distributed through the ventricles and subarachnoid spaces. It has no relation to the perivascular, pericapillary or perineuronal spaces.

Fourth, it possesses no function of and plays no rôle in nutrition. The nutrition of the brain and cord takes place as does that of the other tissues — through its blood vessels, the perivascular spaces playing the same rôle as do the perivascular lymph spaces in the other organs and tissues. The old belief that the brain and cord have no lymphatic system must be abandoned.

Fifth, the cerebrospinal fluid has its source in the choroid plexuses and perhaps in the general serous surfaces of its containing cavities. It leaves the subarachnoid spaces of the cranium by passing through the arachnoidal villi into the venous current of the sinuses; also to a lesser extent by the lymph sheaths of the cranial nerves; from the spinal subarachnoid space it passes out by the lymph sheaths of the spinal nerves.

Sixth, attempts at medication of the brain and cord through the subarachnoid space, as in the Swift-Ellis method, are unscientific, as substances introduced into the cerebrospinal fluid rapidly disappear by passing out through the arachnoidal villi and the lymph spaces without in the slightest degree penetrating the nervous parenchyma; the beneficial effects hitherto ascribed to the Swift-Ellis and kindred methods are due entirely to the incidental spinal drainage.

Seventh, medication of the nervous parenchyma must be attempted through the alimentary tract, through the skin, through the areolar tissue, or directly through the blood.

Eighth, a remedy should be sought the ions of which will readily osmose through the capillary walls.

Ninth, spinal drainage is urgently indicated in tabes and paresis for the reasons enumerated in this paper.

A CASE OF MYXEDEMATOUS PSYCHOSIS

CLINICAL AND PATHOLOGIC REPORT

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INTRODUCTION

In 1873, Gull described five cases of a peculiar disease under the title, "On a Cretinoid State, Supervening in Adult Women," and explained the similarity to sporadic cretinism, which was described by Hilton Fagge. In 1877, Orth reported five similar cases, and noticed especially the condition of the skin, in which he found an excess of mucin. He, accordingly, named the disease myxedema. He found also, in a case that came to necropsy, atrophy of the thyroid gland, which was previously noticed by Curling in a case of sporadic cretinism.

An exact understanding of this condition, however, is dated from the time when Reverdin and Kocher recognized cachexia strumipriva, and when the same condition was brought out experimentally by removing the thyroid.

Semon is the first one who claimed definitely that myxedema, cachexia strumipriva, sporadic and endemic cretinism, are in the same disease group, and are characterized by the failure of the function of the thyroid.

Alcoholism, syphilis and numerous pregnancies, especially when accompanied with much loss of blood, have been thought to be the cause of myxedema. Kocher described a case in which myxedema was accompanied by gummatous syphilis of the thyroid, both being cured by iodin treatment. The same author observed another case in which actinomycosis was the cause of the disease; the patient was cured by incision and drainage. Sometimes myxedema is found in conjunction with goiter. These cases are reported by Murray, Heinzheimer, Gantier, Robertson, Imredy, Hischl, Ulreich and others. Among the most important causes of the disease are also acute infectious diseases, such as typhoid, malaria, acute rheumatism, pneumonia, etc.

Though the disease is found nearly everywhere, it is said to be more frequent in England, France and Switzerland. It occurs in cold climates rather than in the tropics, but it is not common in North America.

Women are far more frequently afflicted than men. Prudden found 145 cases, 32 men and 113 women; Murray, 425 cases, 55 men and 370 women; Heinzheimer, 127 cases, 10 men and 117 women. The predisposition of women is probably due to frequent congestions and toxic infectious disturbances of the thyroid during menstruation, pregnancy, menopause, etc.

The most pronounced symptoms are myxedematous infiltration of the body, dryness of the skin, falling of the hair, slowness of the movements and certain nervous and mental symptoms. Sensory organs are often afflicted; hearing, taste and smell are diminished. Deafness is quite common, and it is thought to be of central origin. The extremities, lips and nose are cold and cyanotic, circulation is slow and the pulse rate varies from 50 to 65. Cerebellar symptoms due to the disease were reported in 1910 and 1911 by Soederbergh.[1] He observed cases with adiadokocinesis, Babinski's cerebellar catalepsy and exaggerated irritability of the muscles, but without symptoms of dementia.

The frequency of myxedematous psychosis is differently reported by various authors. Wagner and Jauregg[2] gave a percentage of 15. In the majority of cases the psychosis occurred late, after many years' duration, but in a few cases the psychosis occurred in the beginning of the disease, as reported by Pilcz, Inglish and Sierau.

The symptoms of the psychosis are not characteristic. Most patients seem to be demented, with more or less delusions, which are not infrequently based on hallucinations. The delusions are apt to be persecutory in character. The tendency to misbelieve is claimed to be one of the characteristics of the disease, and is an important factor in the development of the delusional conditions. Under the influences of persecutory ideas, patients are apt to become disturbed, cry, become agitated and sometimes extremely violent. In other cases there are reported several different symptoms, such as grandiose ideas, manic exaltations, or melancholia, anxiety and suicidal ideas. The latter cases are reported by Pilcz, Kraepelin, Show, Inglish, Beadles, Clouston, Hamilton, McLane and others.

The most important pathologic anatomical finding is that of the thyroid; even by palpation the organ shows remarkable atrophy. At necropsy it weighs one-tenth of the normal gland. Burghart observed

1. Söderbergh: Ueber Pseudotetanus myxoedematoides, Monatschr. f. Psychiat. u. Neurol. **32**:402, 1912. Encore un cas de myxoedème avec symptômes cérébelleux, Rev. neurol. **19**:86, 1911. Faut-il attribuer à une perturbation des fonctions cérébelleuses certains troubles moteurs du myxoedème, Rev. neurol, **2**:487, 1910.

2. Wagner and Jauregg: Myxoedema und Kretinismus, Handbuch der Psychiatrie, Aschaffenburg, 1912.

a case in which he could not demonstrate any gland tissue. Some authors, such as Cunningham, Robinson, Corkhill, Schwass and others, have reported cases of enlarged thyroid in living patients. Microscopic examination shows marked increase of fibrous tissue. Some authors observed lymphocytic infiltration in the parenchyma in the early stage of the disease. According to Prudden, the parenchyma of the gland is more or less completely replaced by fibrous tissue and by newly formed reticular tissue in which are lymphocytes, resembling the tissue of the lymph nodes.

The majority of authors consider that the increase of the connective tissue is secondary, and that the primary cause is the atrophy of the gland tissue, which is probably due to some toxic agent. Ponfick believes in the possibility of a primary infection of the interstitial tissue which may give rise to secondary atrophy of the gland tissue.

The pituitary body is often found enlarged, as in the cases of Boyce and Beadles, Ponfick, Murray and others, but in one case Ponfick found atrophy of the gland. The relation of the pituitary body to myxedema still remains obscure.

No characteristic, regularly appearing findings have been reported in other organs of the body. The pathologic anatomy has given no satisfactory explanation for so marked disturbances in the mental and psychical realms. Brain, spinal cord and nerves are found either normal (Hun, Prudden, West and Harely), or the findings are not absolutely characteristic.

In about half of the cases atheroma of the aorta and large arteries has been found. Endarteritis of the small arteries is also one of the frequent findings.

In the Danvers State Hospital laboratory we have had two cases of myxedema in a total of 2,130 necropsies, beginning in 1879 and ending in the present year. The necropsy of the first case, a 45-year-old female, case number 15840, was performed (Nov. 16, 1910) by Dr. A. H. Peabody.[3] The clinical symptoms and pathologic findings correspond to those of a typical case. But, I regret to say, I could not study that case in connection with the present one, because the brain was not available and the description concerning the central nervous system is missing. Some of the clinical and pathologic symptoms, however, show striking similarity to the present case, and will be referred to later in the discussion. The second case is the present one, of which we have been able to make a thorough study and to report as a typical case of myxedema.

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3. Peabody: An Histological Study of the Thyroid Gland in Mental Disease, with Special Reference to Chronic Thyroiditis, Danvers State Hospital Laboratory Papers, 1910.

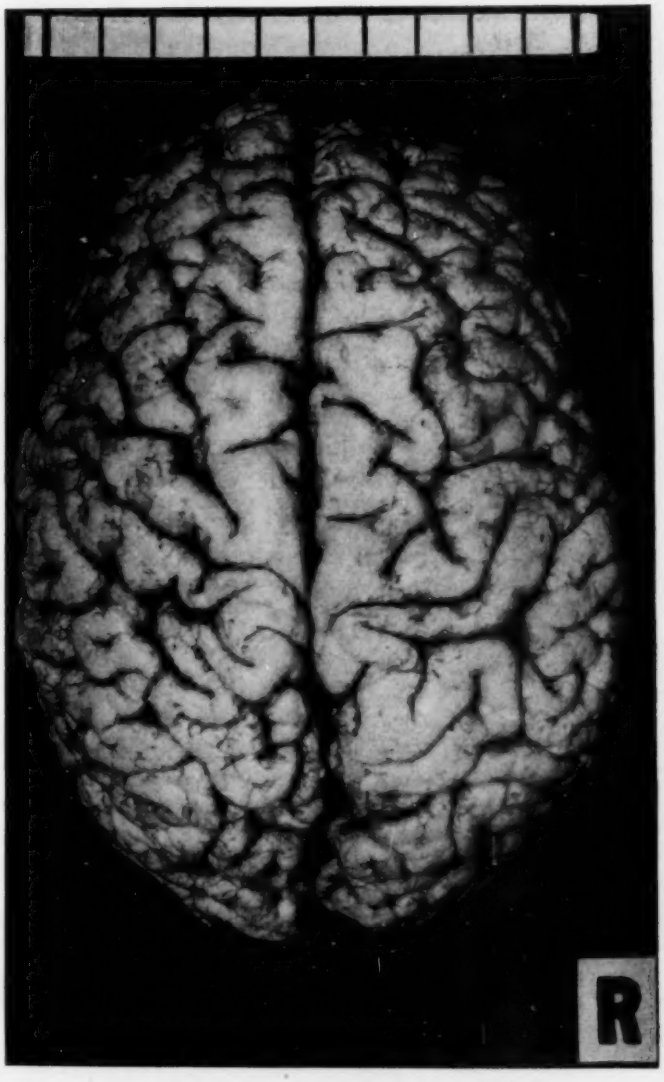

Fig. 1.—Superior surface of the brain.

REPORT OF A CASE

CLINICAL OBSERVATION

History (Danvers State Hospital, Clinical No. 21477).—There was no history of nervous or mental diseases in the family, so far as was known.

The patient was born Jan. 1, 1853, in Nova Scotia. Early development showed nothing abnormal. She had very little education, and did not go to school until she was 9 or 10 years old, and then only for six months. She could barely read and write. She left home at 18 to do housework, and when about 20 married her cousin, by whom she had two children, one of whom died in infancy. Her husband died of typhoid and pneumonia two years after marriage, and shortly after that she came to the United States (about forty years ago), and did general housework. Thirty-eight years ago she married again, and had three children, one of whom died in infancy. The informant stated that the patient had some pelvic trouble ten years ago when about 55 years of age, and was operated on for two large broad ligament cysts on the right side; at the same time her appendix was removed.

Since that time she had been gradually failing, both mentally and physically. Her memory, especially for recent events, began to fail. She had had many attacks of vertigo, sometimes falling to the floor. These symptoms have gradually been growing worse, especially during the past year. About a year ago she was in danger of losing her home, and her son took her to his home and tried to keep her with him. She had always been very stubborn, self-willed and dictatorial, and insisted on having her own way. He could do nothing with her, and during the past year her daughter has been taking care of her. She became very untidy, and would wander about on the streets attired very carelessly. She persisted in going back to her own home. She would put things away, lose them, and then accuse her daughter of stealing them. At times she seemed depressed. She threatened to commit suicide, and on one occasion she tried to turn on the gas. Her daughter was afraid to leave her alone in the house. She would meddle with the cooking, and spoil the food by putting salt in it; she was dull and stupid, sleeping a good deal during the day. Her appetite was capricious; sometimes she ate an enormous amount. She was admitted to the Danvers State Hospital, March 24, 1919.

Physical Examination.—General: Her height was 4 feet 8 inches; her weight, 130 pounds. She was well developed and well nourished. The skin over her face and neck was pale yellowish and waxy. The musculature showed poor development, not much tone. Edema of the face, arms and legs was pronounced. The hair was grayish and very sparse; she was bald over the vertex. There was a linear scar extending from the umbilicus to the pubic bone.

Thoracic, Respiratory and Circulatory Organs: Results of the examination were negative except that respiration was slow, 14 per minute, and the pulse rate was only 60 per minute. The body temperature on admission was 98.8 F.

Digestive and Abdominal Organs: Teeth were missing in the upper jaw, and a few stumps remained in the lower jaw. The breath was fetid. The tongue was extremely large and flabby and was slightly coated. The abdomen was large and pendulous.

Genito-Urinary Organs: No internal examination was made. The urine (March 27, 1919) was cloudy and straw colored. The specific gravity was 1.026. It was acid and showed a trace of albumin, mucous threads, epithelial cells, a few leukocytes and a few oxalate crystals, but no sugar. The Wassermann reaction on the blood serum was negative.

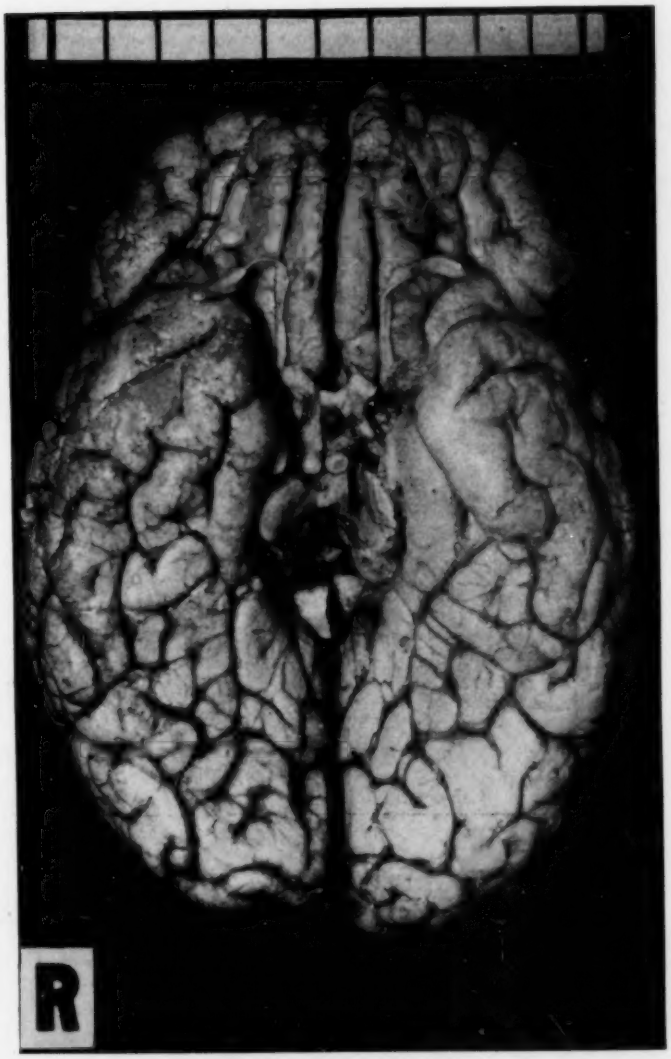

Fig. 2.—Inferior surface of the brain.

Nervous System: General Sensations: She said that she had a feeling of weariness and exhaustion and wanted to sleep all the time. When she arose she was extremely dizzy and sometimes fell.

Eyes: The eyelids were so puffy that her eyes were almost invisible. She complained that her vision was poor; the pupils were unequal, the right being smaller, and they were very sluggish in responding to light.

Ears: She was totally deaf in the right ear, and could only hear when one shouted in the left ear.

Taste and Smell: These were apparently normal.

Cutaneous Sensibility: Tactile appreciation was somewhat impaired. She found it difficult to differentiate between pin prick and pencil. She was not always able to localize for time and space. She complained of numbness of the left arm and leg.

Deep Sensibility: There was no tenderness of the nerve trunk on pressure. The stereognostic sense was good.

Vasomotor Condition: There was an almost general edema, more pronounced in the face, hands and feet, and considerable abdominal ascites.

Reflexes: The knee jerks were sluggish, especially in the left knee. There was no ankle clonus. The organic reflexes were impaired.

Motor Functions: Motility of the facial muscles was poor, owing to the general edema. The tongue protruded in the median line. There was no tremor. The gait was unsteady. Coordination of movements was poor. She was unable to balance herself without assistance, and could not stand in the Romberg position. She had fallen several times, and said this was due to dizziness. She slept all night and the greater part of the day.

Mental Examination.—General Appearance and Attitude: Her face was so waxy and expressionless that with a little "touching up" her resemblance to a clown would be striking. She lay in bed in a sort of stupor most of the time.

Speech: Speech was thick, and she talked in a harsh, raucous voice.

Consciousness and Orientation: She was partially oriented. She knew that she was at Danvers, but was not oriented for time and person. She said it was 1993.

School Knowledge: This was very meager.

Calculation Ability: Her ability to calculate was very poor.

Hallucinations: She had had no hallucinations so far as could be ascertained, and there was no history of them.

Memory: Her memory for remote events was fairly good, but poor for recent events.

Judgment: It was impossible to elicit any definite delusions. She said she did not get along very well with her husband, but denied that it was her fault. She said that everybody tried to boss her around, and she could not stand it any longer. She admitted that she was a little forgetful, but she could remember what happened twenty years ago as well as any one. She was lacking in insight.

Emotions: She was not at all emotional; in fact, she was dull and apathetic. She showed no apprehensiveness, and had not been irritable since she came to the hospital.

Social Relations: She was untidy in her habits, but otherwise caused very little trouble.

Course of the Disease.—March 27, 1919: Slept most of the time. Rather stupid; inordinate appetite.

April 4: She was stupid and sleepy; very deaf; disoriented; no delusions.

April 22: She failed gradually and died today.

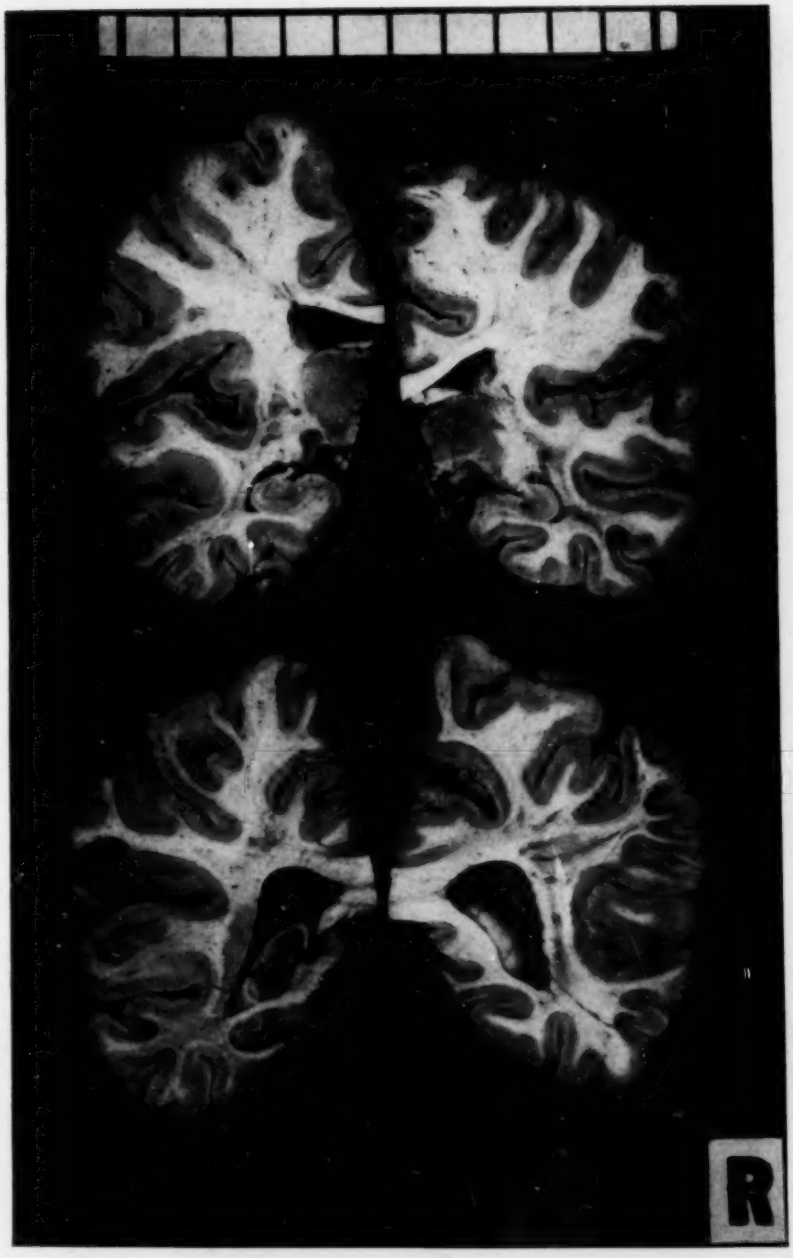

Fig. 3.—Cut surfaces of the brain.

POSTMORTEM EXAMINATION

(Made twenty hours after death)

1. *Gross Anatomy.*—General Description: The body weighed 57 kg.; its length was 150 cm. The body was that of a well developed and well nourished female, aged 66, but apparently older. The skin over the face and neck was yellowish; other parts were grayish white. The face, chest, upper and lower extremities showed edema. There was rigor mortis. The hair was grayish and very sparse, and the head was bald over the vertex. The face was waxy and reminded one of a clown's face. The eyelids were swollen and edematous; the palpebral and orbital conjunctivae were edematous and injected; the pupils were slightly irregular; both measured 4 mm. The nose was broad and swollen, but otherwise not remarkable. The teeth were poor; many were missing. The tongue was thick and coated. There was no discharge from the ears. There was a small nevus in the submental region. The thyroid was not palpable. The chest was well formed. The abdomen was flabby and pendulous. There was a scar extending from the umbilicus to the pubic bone. There were marked striae. There was an exostosis over the upper part of the right tibia. The lymph nodes were not palpable.

Ventral Section: The fat over the chest measured 1 cm., over the abdomen, 2.6 cm., and was very pale in color and of fluid-like consistency. The muscles were pale in color, and were also edematous. The omentum was folded back over the transverse colon, and was rich in fat. The stomach was markedly distended. The appendix was removed; also the right ovary. The uterus was in midline. There were no adhesions around the gallbladder. The spleen was free.

Thoracic Cavity: The mammary vessels stood open on section. The lungs did not meet and were retracted. There were no adhesions around the lungs. The pleural cavities contained free fluid. The pericardial sac was enormously distended, and contained an increased amount of straw-colored fluid.

Heart: It weighed 470 gm. and measured 12 by 13 by 6 cm. The descending branch of the left coronary artery was remarkably sclerosed, and a part of it practically occluded by yellowish atheromatous softening. The measurements were: tricuspid valve, 11.5 cm.; pulmonary valve, 8 cm.; mitral valve, 9.5 cm. The mitral valves were calcified and contracted. The ostium admitted more than three fingers. The aortic valve measured 7 cm. The cusps were thickened. Origins of the coronaries were remarkably calcified. The left ventricle wall measured 2.2 cm., the right ventricle wall, 0.6 cm. The cardiac muscle was pale and soft.

Left Lung: The left lung weighed 400 gm. and measured 22 by 16 by 4 cm. There was no apical scar. The anterior edge of the upper lobe was emphysematous. The surface of the lower lobe was grayish in color. The cut surface was dark red; dark colored blood oozed from the lung; there was no frothy fluid. Sections floated. The bronchial wall was injected and covered by hemorrhagic fibrinous exudation. The peribronchial lymph nodes were enlarged and pigmented.

Right Lung: It weighed 480 gm. and measured 21 by 18 by 5 cm. The anterior border of the middle lobe was emphysematous. There were hardened areas in the lowest lobe. The cut surface showed an irregular, slightly elevated induration around the bronchi. The bronchial wall was markedly injected. On pressure, bloody, frothy fluid oozed from the lung. Sections floated.

Liver: This organ weighed 1,430 gm., and measured 24 by 19 by 7 cm. The capsule was thickened and slightly opaque. The surface was slightly granular. The color was partly greenish blue and partly reddish. The cut surface showed slight evidence of congestion and fatty change. The gallbladder contained small, sandy stones which were dark in color and crumbled easily. The wall of the gallbladder was injected.

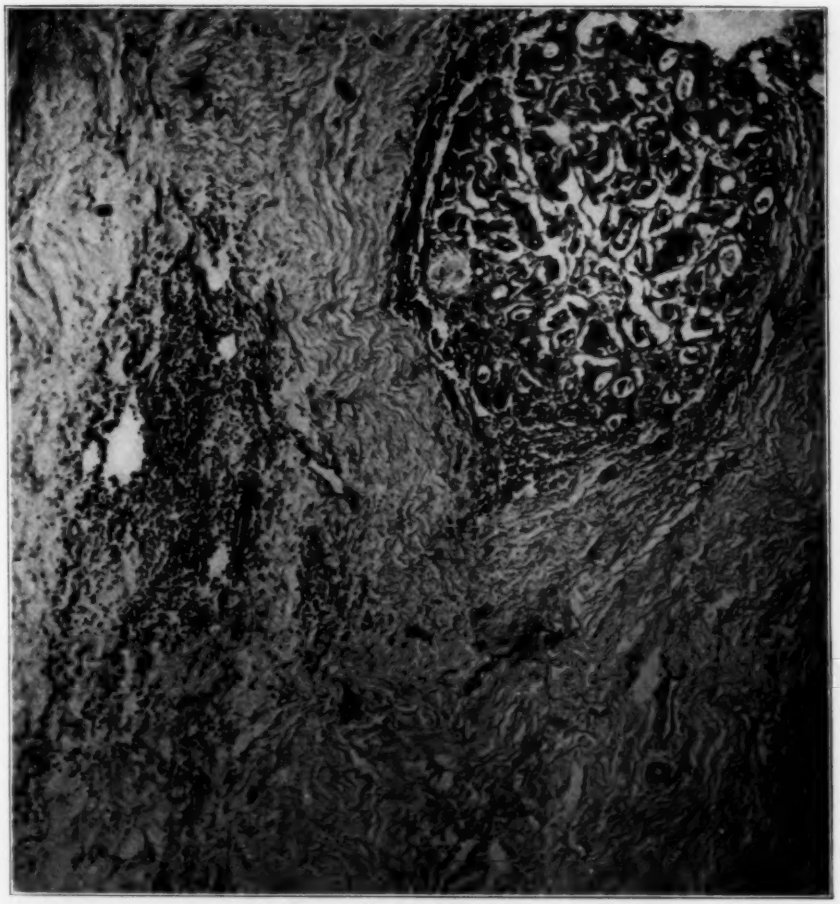

Fig. 4.—The thyroid gland, showing fibrosis and lymphatic cell infiltration. Hematoxylin-eosin staining.

Spleen: It weighed 170 gm. and measured 12 by 8 by 4 cm. It was pear shaped. The capsule was thickened. The surface was granular and clouded. The organ was of unusually hard consistency and there were four fetal lobulations. The cut surface was dark red. The pulp did not bulge and was not easily scraped away. The malpighian bodies were hardly visible.

Left Kidney: This kidney weighed 110 gm. and measured 9.5 by 5 by 3.5 cm. The fatty capsule was increased. The fibrous capsule stripped with some difficulty. The surface of the organ was markedly granular. There were many

small cortical cysts. The cut surface was pale in color. The cortex measured 0.4 cm. The pyramids were not well defined and there were whitish striations in the pyramids which suggested increased interstitial tissue. The pelvic fat was remarkably increased.

Right Kidney: The right kidney weighed 110 gm. and measured 10 by 6.5 by 3 cm., otherwise the description was the same as for the other kidney.

Suprarenals: The cut surface showed some increased yellow deposits.

Thyroid: The thyroid was very small; it weighed 10 gm. Careful dissection revealed no isthmus. The cut surface showed no proper structure of the thyroid gland. It appeared to be represented by some fatty and more fibrous tissue. It was fairly firm in consistency.

Parathyroid: The parathyroid glands could not be identified.

Genito-Urinary Tract: The bladder was contracted and contained no fluid. The mucous surface was not remarkable. The vagina was smooth. There were small hemorrhagic areas. The uterus contained turbid fluid; the wall was hemorrhagic and firm in consistency. Section showed abundance of vessels and increase of fibrous tissue. The right ovary was absent, and the broad ligament was adherent to the stump of removed appendix; the left ovary was smaller than normal. The surface was greatly roughened and firm in consistency. Cut surface showed dilated vessels and small cysts.

Gastro-Intestinal Tract: The stomach was distended and contained a greenish mucoid fluid. Mucous membrane was injected. The intestines contained a chocolate colored fluid. The intestinal walls showed a bluish green discoloration, probably of postmortem change. The colon and rectum were not remarkable.

Pancreas: Nothing remarkable was found in the pancreas.

Head: The scalp was thick and adherent to a dense calvarium. There was no diploe. The calvarium was somewhat thickened. The grooves for the middle meningeal arteries were not deep.

Brain: The brain weighed 1,000 gm. The dura mater was thickened. The pia mater was irregularly thickened and showed milky cloudiness over the vertex. The brain was soft and edematous. Superior surface of the brain: There was congestion of the veins, especially on the left side. The pia mater was adherent everywhere over the lateral surface of both hemispheres. A large part of the right posterior central convolution was remarkably narrow. Convolutions of the right cuneus, especially bordering on the margin of the hemisphere, appear much atrophied. The posterior part of the right lingular gyrus was markedly sunken from the surface. The upper part of the left posterior central convolution and left superior parietal convolution was much narrower than normal. The angular gyrus of the left side was also atrophic. Base of the brain: The cranial nerves were not remarkable. The basilar artery and internal carotids were remarkably sclerotic. The pons appeared smaller than normal and resistant to the touch.

Cut surfaces of the brain (Meynert's section): Nothing remarkable was found, except that the above mentioned atrophic convolutions appeared also atrophic on section.

Pituitary Body: This was slightly smaller than normal and was firm in consistency.

Internal Ears: Nothing of note was found in regard to the internal ears.

Spinal Cord: Nothing remarkable was found in the spinal cord.

Anatomic Diagnosis: The body was well developed and well nourished, and there was myxedema of the entire body. The fingers were cyanosed. The

conditions noted included alopecia, a waxy clown-like face, irregular pupils, poor teeth, thick tongue, operation scar on the abdomen, exostosis over the upper part of the right tibia, myxedematous condition of the muscle and fatty tissues, ascites, the appendix and the right ovary missing (by operation). There were also hydrothorax, hydropericardium, dilatation and hypertrophy of the heart, arteriosclerosis of the aorta, major arteries of the body and coronaries; also basilar and cerebral arteriosclerosis; increased pericardial

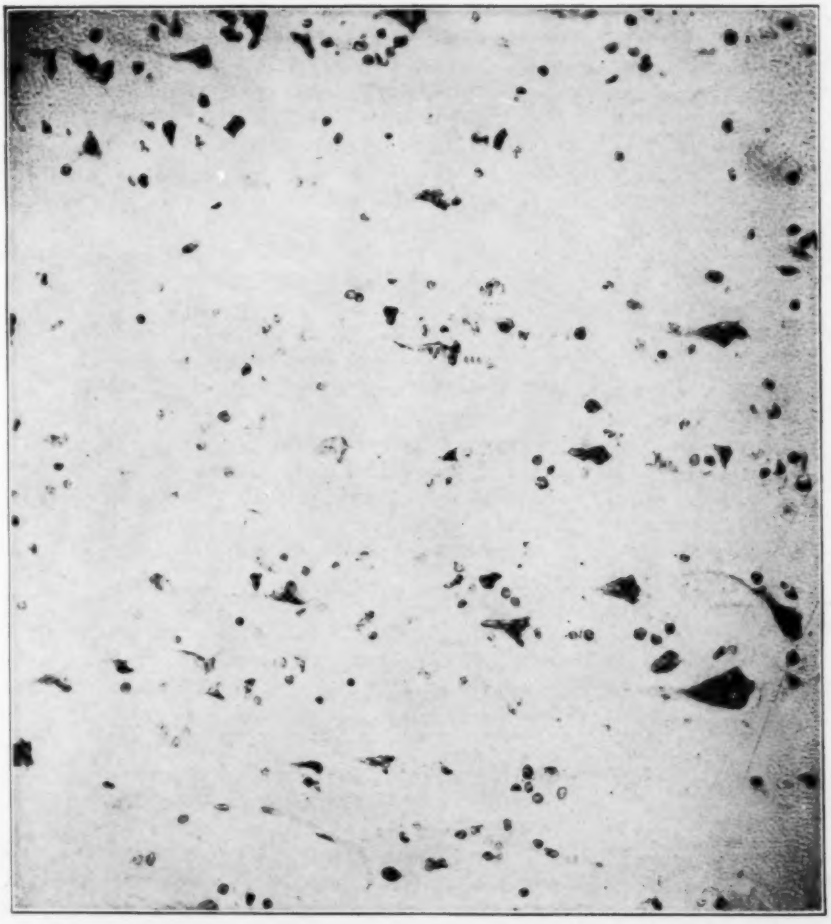

Fig. 5.—Cell sclerosis with fatty degeneration, cells of third layer, superior frontal convolution. Thionin staining.

fat, emphysema of the lungs, hemorrhagic fibrinous bronchitis, bronchopneumonia of the right lower lobe, congestion and fatty change of the liver, cholelithiasis, a large spleen with increased trabeculation, fatty and fibrous replacement of the thyroid, chronic endometritis and eburnation of the calvarium. Diploe was absent. The brain was small, weighing 1,000 gm. There were chronic pachymeningitis, chronic leptomeningitis and irregular atrophy of the convolutions.

2. *Microscopic Anatomy.*—(*a*) General Histologic: Thyroid: This was almost completely replaced by fibrous tissue. The ordinary tissue was extremely atrophied, appearing here and there like tiny islands. The thyroid contained practically no colloid matter. The remaining gland tissue was infiltrated and surrounded by small spheroidal or lymphatic cells. In some parts the small lymphatic cells occupied the place of the gland tissue and gave the appearance of the normal lymphatic tissue of the lymph nodes. The small arteries, both in the newly formed lymphatic tissue and in the connective tissue, were remarkably thickened, but showed no perivascular infiltration.

Pituitary Body: The anterior lobe was somewhat smaller than normal and the posterior lobe was remarkably small. The cells of the anterior lobe appeared rather numerous and compact, and the acidophilic cells had increased in the central part as well as in the periphery. The intermediate part extended further into the posterior lobe, but showed no evidence of secretion of colloidal matter. There was slight thickening of the walls of the vessels, and they were infiltrated by lymphocytes, differing in this respect from the vessel picture in the thyroid.

Uterine Wall: There was a formation of a thick layer of newly formed, very vascular tissue over the surface of the mucous membrane.

Ovary: There was a formation of dense new interstitial connective tissue around the walls of the arteries and the surface of the organs. The veins were dilated. Here and there obliterating endarteritis appeared.

Adrenals: These showed a moderate grade of fatty degeneration.

Pancreas: Nothing remarkable was found in the pancreas.

Spleen: Chronic indurative splenitis with abnormally increased pigmentation (hemosiderin) in the cells of the pulp was found.

Lung: The right lower lobe showed exudative pneumonia about the bronchial tubes forming the lobular areas.

Heart: There was brown atrophy of the cardiac muscle with areas of scar formation.

Liver: Chronic congestion and fatty change were found.

(*b*) Microscopical Findings of the Brain: Pieces were taken from the superior frontal, anterior and posterior central, Heschl's transverse and superior parietal convolutions of both hemispheres; also calcarine region, cornu ammonis and several other parts appearing atrophic by macroscopic observation. These pieces were cut by frozen section and by embedding in paraffin, the latter for the purpose of staining by thionin, hematoxylin-eosin, Van Gieson and glia method, the former for fat staining, modified Weigert-Pal's staining and Bielschowsky's silver impregnation.

Hematoxylin-Eosin and Van Gieson's Method: The pia mater was irregularly thickened, especially over parts of convolutions which appeared atrophic. The vessels of the pia mater and upper cortex layer showed marked arteriosclerotic alterations. The thickened parts of the pia mater and the cortex underneath, which often had sunk from the surface, contained numerous amyloid corpuscles. Here and there walls of vessels were calcified. Small vessels appeared to be much increased, disturbing the cell order of the cortex. The width of the cortex was irregular. In highly atrophic parts, such as those mentioned above, and in the neighborhood of the thickened vessels coming down from pia mater, the ganglion cells of the cortex had entirely disappeared, glia cells and glia fibers occupying such areas. The vessels in the deeper layer of the cortex and in the white matter were also thickened and showed marked perivascular gliosis.

Thionin Staining: There was marked disorder of the cells in the superficial cell layers, especially in those of the frontal, anterior central and posterior central convolutions of both sides, right cuneus, posterior part of the right lingular convolution and left angular gyrus. In the deeper parts of the cortex the cell order was fairly regular, although there was some deviation of apical dendrites and some slight disarrangement of the cells. This condition suggested that the cortex layers supplied by short pial vessels had suffered more than the deeper cell layers which were nourished by medullary branches.

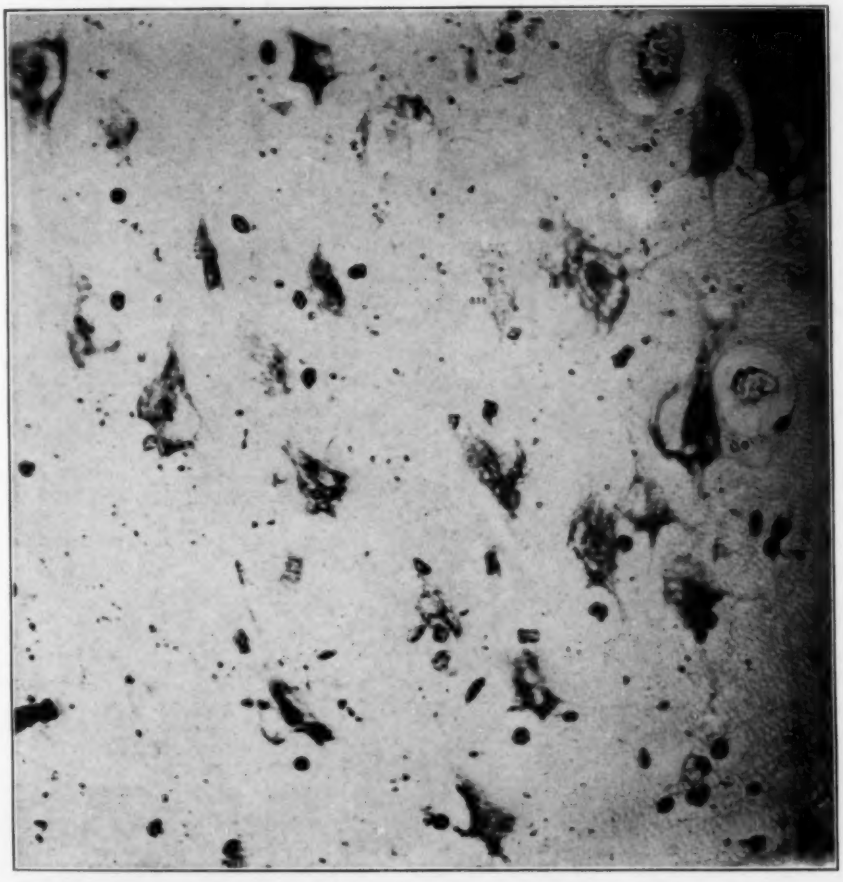

Fig. 6.—Ganglion cells of cornu ammonis showing cell change associated with edematous condition of the brain. Thionin staining.

The cell changes can be divided roughly into three different groups:

1. *Cell Sclerosis (Nissl*[4]*) Combined with Fatty Degeneration (Fig. 5).*—This change was found in almost all of the cells of the second and third layers

4. Nissl: Der gegenwärtige staud du Nervenzellenanatomie und Pathologie, Allg. Ztschr. f. Psychiat. **51**:981, 1894-1895.

(Broadmann) and the majority of the deeper layers. These sclerotic cells showed dark stained nuclei often of elongated, pyramidal or polygonal shape. The protoplasmic body was also darkly stained and remarkably shrunken, making the border between protoplasm and nucleus obscure. The apical dendrites were well stained and traceable to a greater distance than in normal cells. Most of the apical dendrites of the second and third layers took a winding course. These sclerotic cells carried on their side or on their base protoplasmic reticulum which contained greenish and yellowish looking lipoid masses. A few of the apical dendrites contained also a lypoid mass and appeared to be swollen. This fatty sclerotic degeneration was found more markedly in atrophic parts of the cortex than in other places.

2. *Fatty Degeneration with Granular or Heavy Degeneration.*—The cells of the deeper layers (Figs. 5 and 6) and the large cells of the third layer contained a remarkably large amount of fatty pigment. The protoplasmic reticulum containing fatty pigment was located either on the base, the side of the cell or over the apex. The nuclei of these cells were stained a little deeper than normal showing a wheel-like structure or a nebulous, less distinct structure around the nucleus. The nucleus was located eccentrically, owing to the pressure of the deposited fatty substance. It was irregular in most of the cells. The membrane of the nucleus was more or less preserved. Nissl's granules in these cells were either in good condition, finely granulated or had disappeared. Betz' cells, the large cells of the post central region, superior temporal convolutions and calcarine region showed a fairly good condition of Nissl's bodies. In the cells of the other parts granular degeneration predominated or the cells showed dark stained protoplasm with a net-like structure in it.

3. *Cell Change Associated with the Edematous Condition of the Brain* (*Fig. 6*).—This kind of cell change is found both in the superficial and in the deeper layers, combined with other cell degenerations. Nissl is the first author who gave attention to this condition of the cells. The protoplasmic part was torn apart from the nucleus or from the protoplasm around the nucleus. In some cells a crevice was observed across the width of the protoplasm or a vacuole-like cavity in several parts of the cell, some in the base or sides, others in the apex or even in the apical prolongation. The cavities were irregular in shape and appeared to be made by pulling apart a portion of the cell. At first these cavities were thought to be the remains of fat globules which had been dissolved by alcohol, but it was found that the same cavities were present in formaldehyde fixed specimens stained with scarlet red without the use of fat solvents. In the cornu ammonis the same condition of cavity formation was found around the nucleus showing a wide space between the protoplasm and nucleus. This kind of cell change is said to occur in cases in which the brain is found to be soft and edematous at necropsy. It is possible to cause this kind of cell change by the use of the reagents which withdraw water instantly from the brain tissue. Simchowicz[5] says: "These cell changes are artefact to a certain extent, inasmuch as they are caused by fixing, but they may also be considered pathological, as their formation under fixation is the result of a definite pathologic condition, i. e., an edema of the brain."

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5. Simchowicz: Histologische Studien über die senile Demenz, Histologische und histopathologische Arbeiten über de Grosshirninde, Nissl, 1911.

In our cases the brain was taken out twenty hours postmortem. Before fixing in formaldehyde it was soft and edematous. We could not, of course, be sure how great a rôle the postmortem change played in this condition. The body, however, was kept cold (25-30 F.) and the weather at the time was very cool, and other brains coming to necropsy under the same conditions showed no such change. Moreover, the skin, fatty tissue and the muscles

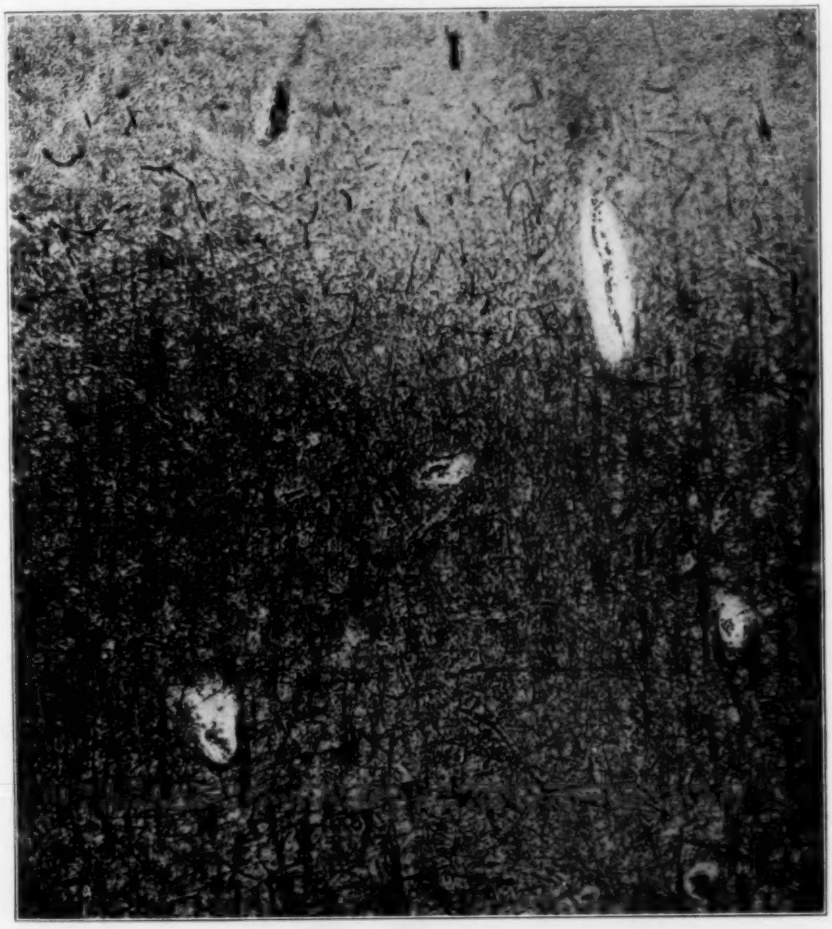

Fig. 7.—Degeneration of tangential fibers and supraradiate cross striations. Weigert-Pal myelin sheath staining.

showed edematous condition due to the disease. There is no reason, therefore, why the edematous condition of the brain should be exempted from the pathologic change due to the myxedematous condition. Admitting that this cell change is partly artefact, caused by fixing solution, it must still be regarded as pathologic because of the body disease and the condition of the brain, which gives rise to this change.

Bielschowsky's Staining: This staining showed very plainly the alterations of small vessels, especially the so-called arterio-fibrosis of Friedmann, which Simchowicz and Fuller[6] have illustrated in their papers and called attention to as one of the essential findings of senile changes. The arteriofibrosis was found more markedly in vessels of the upper cortex layers. Degeneration of the cortex beneath the pial covering and the scar formation around the arteriosclerotic vessels were observed. Sclerotic cells with fatty degeneration as compared with Nissl's staining showed dark stained protoplasm and nuclei with elongated apical dendrites. Intracellular and extracellular neurofibrils showed segmentation. Larger cells of the deeper cortex layer possessed pale stained nuclei and fairly well preserved intracellular neurofibrils. Fatty degeneration and cell changes caused by the edematous condition of the brain tissue were also observed.

Scarlet Red Staining: Fairly abundant fat corpuscle cells were found in thickened parts of the pia mater. The walls of the vessels of the pia mater and of the brain showed fatty degeneration. Fat corpuscle cells were found around the vessels and the highly degenerated cortex parts. Fatty degeneration of the ganglion cells is one of the most striking features of this case. All cells of the upper and the deeper cortex layers showed a deposit of an extremely large amount of fatty pigment substance. This condition existed over the entire brain tissue, while in the degenerated cortex and in the cornu ammonis it was perhaps most prominent. Spider cells of the surface layer and the large pale stained nucleus of the glia cells showed marked fatty degeneration.

Myelin Sheath Staining (Fig. 7): Tangential fibers were to a marked degree destroyed, here and there showing the remains of fine fibers and some hypertrophic ones. In the highly degenerated cortex the myelin sheaths are lost. Supraradiate cross striations (Supraradiaere Flechtwerke) were scarce. In the white matter there were degenerations running longitudinally with the vessels.

Glia: The glia cells were increased, especially in the deeper parts of the cortex and the white matter. Small dark stained nuclei were not so numerous as pale stained, larger forms with large nuclei which were often stained metachromatically. Around these were often observed (by thionin staining) yellowish and greenish appearing lipoid granules which stained markedly with sudan or scarlet red. This condition of lipoid accumulation around the nuclei was seen more distinctly in the cortex than in the white matter. In the neighborhood of the vessels of the white matter and the cortex, glia cells were remarkably increased.

Weigert's Glia Staining (Fig. 8): The surface glia layer was distinctly broader than normal, some of the fibers reaching deep into the cortex. The glia fibers were also increased remarkably in the atrophic parts of the cortex and around the thickened vessels. On the cortex surface where the atrophic parts had sunk from the normal level, the glia fibers were denser and penetrated deeper into the cortex. The whole feature of this picture of increased glia fibers is like a wedge form. The glia fibers consisted of a small number of thick fibers and a large number of finer and delicate ones. There were also areas in which glia fibers were growing into the pia mater, deep into the pial tissue. Corpora amylacea were very abundant in the atrophic parts of the cortex and the pia mater, where the glia fiber proliferation was most prominent.

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6. Fuller: Further Observations on Alzheimer's Disease, Proceedings of the American Medico-Psychological Association, Sixty-Eighth Annual Meeting, 1912.

(*c*) Microscopic Examination of the Cerebellum: Scarlet Red Staining for Fatty Degeneration: Purkinje cells stained diffuse brownish, showed no fat corpuscles or granules. Dendrites were not visible. There was a small number of fat corpuscle cells in the Purkinje cell layer. Walls of capillaries and arteries showed fatty degeneration and a small number of fat corpuscle cells were seen around them. There were a few corpora amylacea in the deeper part of the molecular layer and Purkinje cell layer.

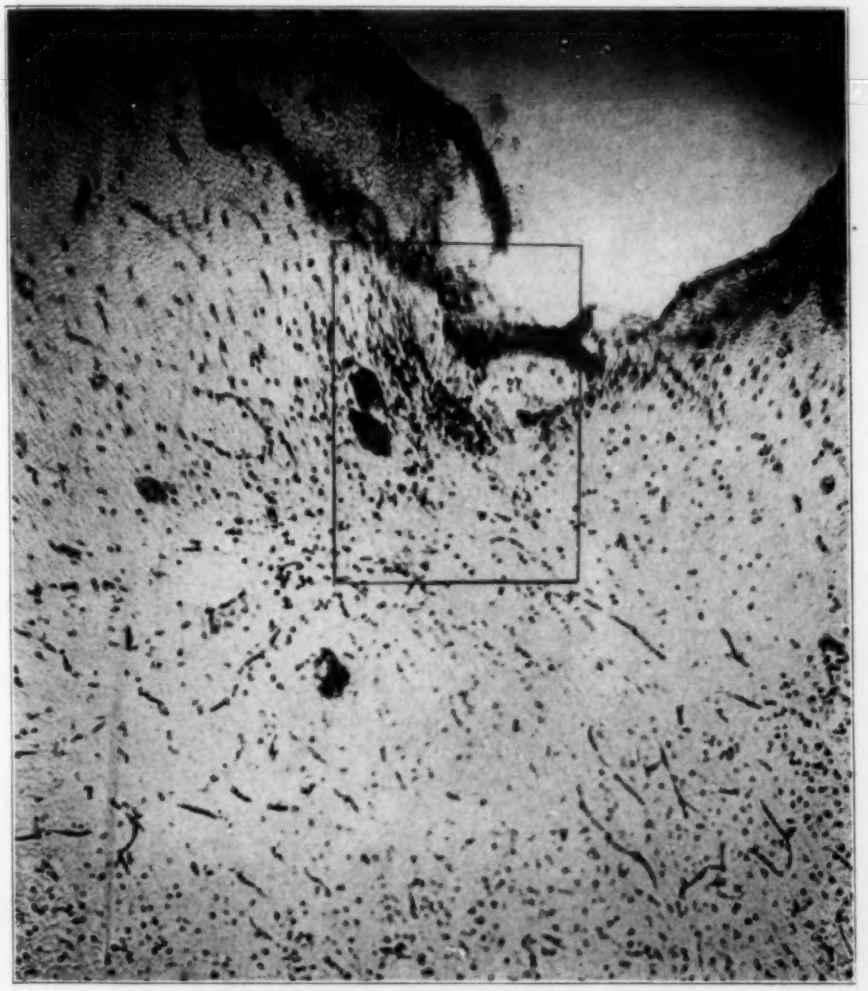

Fig. 8.—Thickening of the surface glia layer, thickening of the walls of vessels, luxuriant growth of the glia fibers and accumulation of amyloid corpuscles. Weigert's glia staining.

Bielschowsky's Staining for Neurofibrils (Fig. 5): The molecular layer was reduced in width. The cells in this layer, both those in the upper part and those in the deeper zone, were remarkably decreased. Tangential fibers showed

also diminution and they took a wavy course. The Purkinje cells were markedly destroyed, and those remaining appeared smaller and poorly stained, though an epicellular and endocellular network existed. Dendrites were for the most part absent, so that the molecular layer appeared much lighter; basket fibers and cushion fibers were diminished. The granular layer also appeared paler on account of the scarcity of the small spheroidal ganglion cells. Remarkably swollen fibers, whose origin was hardly determined, were found both in the molecular and granular layers. They might be either basket fibers, afferent fibers or nerve prolongations of Cajal's cells of the granular layer.

Some of the axis cylinders were swollen (not far from the Purkinje cells) to a peculiar shape, spindle, spheric, sac form or other shape. Neurofibrils in this swollen body appeared mostly homogeneous, taking diffusely silver stain. This latter finding is the same as that described and explained first by Cajal and later by Rossi, Marinesco[7] and Schaffer.[8] These authors found this peculiar local swelling of axis cylinders in some special diseases, and claimed that they were the result of a regenerative process of the axis cylinder, the continuity of which is disturbed. I have seen this peculiar swelling of the axis cylinder in several kinds of brain disease, and I am rather of the opinion that this is a kind of regenerative-degenerative process, seen in many kinds of diseases in which there is degeneration of the Purkinje cells. Accurate description and explanation of this change is not the purpose of this paper, but will be given in a later communication. The vessels appeared to be very much increased in number in all the cortex layers and in the white matter of the cerebellum. Each capillary showed marked arteriofibrosis, probably more marked than in the cerebrum.

(*d*) Microscopic Examination of the Spinal Cord: Little of note was found with the exception of a slight degree of arteriosclerosis and fatty degeneration of the cells of the anterior horn.

THE AUTHOR'S CASE COMPARED WITH CASES IN LITERATURE

The onset of the myxedema in this case is unknown. It appears to have begun ten years ago when the patient was operated on for broad ligament cysts. Since that time the patient had been slowly but progressively failing, both mentally and physically. Her memory had begun to fail, especially for recent events. She had had many attacks of vertigo, sometimes falling to the floor, and this condition had become much more conspicuous in the later stages of the disease; she could not stand in the Romberg position, her gait became unsteady, and she showed marked disturbances of coordination movements. The latter symptoms remind one of the cerebellar symptoms of the myxedematous patient, which has been reported by Soderbergh, though accurate examination for these symptoms was not made in the present case. In recent years she had become untidy and would wander about on the streets. At times she became depressed and threatened suicide.

7. Marinesco: Nouvelles contributions à l'étude de la régénérescence des fibres du système nerveux central, Jour. f. Neurol. u. Psychol. No. 17, 116, 1910.

8. Schaffer: Zum normalen und pathologischen Fibrillenbau der Kleinhirnrinde, Allg. Ztschr. f. d. ges Neurol. u. Psychiat. **21**:1, 1914.

Cases like this have also been described by Pilcz and others. Later, before and after her admission to the hospital, she became very dull and stupid, sleeping all the time.

The physical symptoms were typical. There was edema over the whole body. Bald head, "clown-like" face, thick flabby tongue, poor condition of the teeth, deafness, hydrops of the serous cavities, cyanosis of the hands, diminished activity of the reflexes, arteriosclerosis, bradycardia, etc., all correspond to the typical case.

Fig. 9.—Swelling of the axis cylinder of the Purkinje cell (middle), emaciated cell body of the same cell. Bielschowsky silver impregnation method.

Gross anatomy showed an extremely atrophic thyroid gland, both sides equally small, and with no isthmus. In our first case in the Danvers State Hospital series, according to the description of Dr. Peabody, the thyroid consisted of two small, tough, fibrous masses not connected by any isthmus, lying one on either side of the cricoid cartilage and beginning trachea. On section it looked like pale fatty tissue with

much connective tissue. The parathyroid in that case, as well as in the present case, could not be identified. Although the absence of the isthmus is not uncommon, it certainly is not typical. It makes me doubt, therefore, whether there is any relationship between congenital malformation and this disease.

Necropsy revealed the oöphorectomy of the right side and the appendicectomy. It is very interesting to have found the same condition in our former case about which the necropsy protocol says: "Appendix is absent. No tube and ovary on the right. The left tube is invisible in the mass of adhesions. The left ovary is small (residual), and there are thick fibrous adhesions between the ovary and the adjoining tissues and to the ileum." This fact, found both in our former and present cases, suggests to me a probable etiologic relationship between the pathologic condition of ovaries and the myxedema.

Microscopic examination revealed extreme atrophy of the thyroid gland replaced by fibrous tissue and infiltrated by lymphatic cells. The pituitary body was a little smaller than normal, contrary to the statements of authors such as Boyce and Beadles, Ponfick and Murray, who found enlargement of the pituitary body in this disease. Arteriosclerosis of remarkable degree was found in all vessels of the body and the brain. In our former case there was also marked arteriosclerosis throughout the aorta.

As for the brain, cerebellum and spinal cord, we found more or less remarkable pathologic changes differing from the description of some authors such as Hun, Prudden, West and Harely. But are these pathologic findings specific or characteristic of this disease? This is an important question.

Before answering this question, let us survey the nature of these pathologic changes. They can be divided into two groups, the one being more or less localized and the other general in character. The first group is expressed by localized degeneration in certain convolutions, degenerations around the sclerotic vessels of the superficial cortex layers, perivascular gliosis, perivascular and focal accumulation of fat corpuscle cells, destruction of myelin sheaths, proliferation of glia fibers in degenerated parts, irregular thickening of the pia mater and the surface glia layer, etc. Vessels of the pia mater and the superficial cortex layers showed more marked sclerosis than those in deeper layers. The same condition obtained in the arteriofibrosis of small vessels. This group of changes, however, is nothing but the arteriosclerotic alteration of the brain. The second group consists of universal changes, such as fatty degeneration with granular and heavy change (Nissl), cell sclerosis with fatty degeneration, and the cell

vacuoles associated with the edematous condition of the brain. Though the fatty degeneration and cell sclerosis are found in the arteriosclerotic nature of the alteration, they are not universally distributed over the whole of the brain. According to Simchowicz, "Pathologically

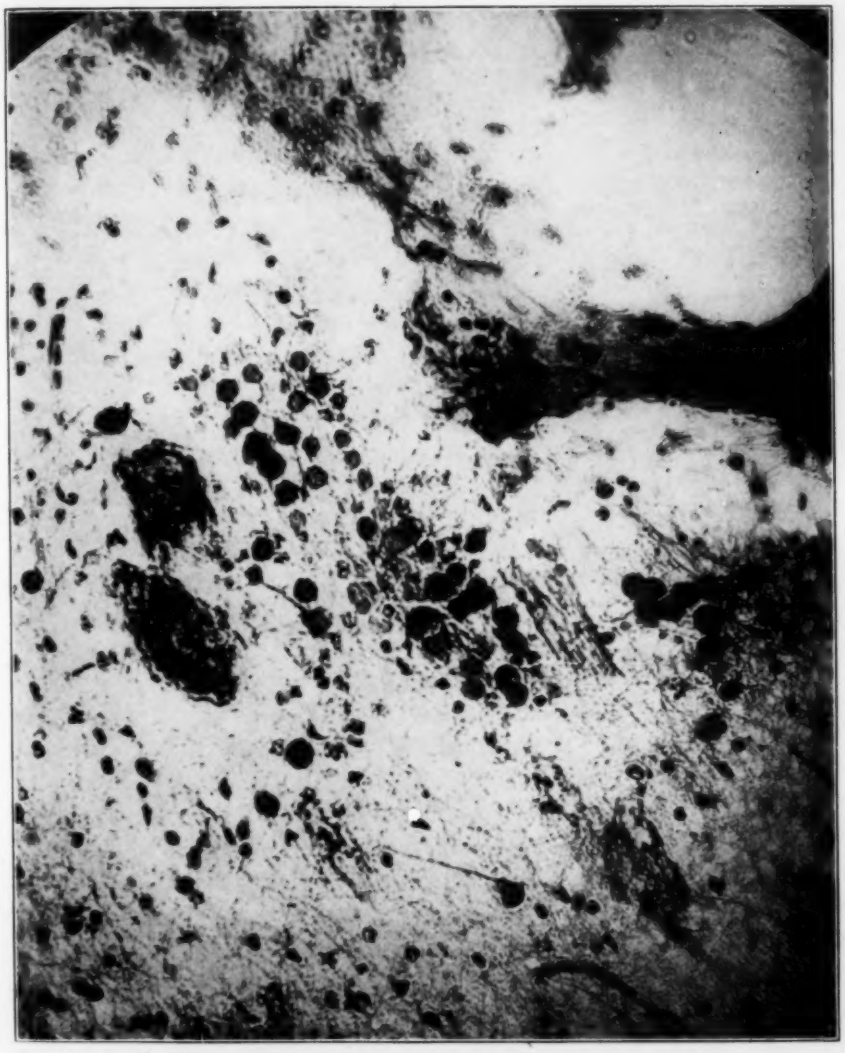

Fig. 10.—Enlarged picture from section of Figure 8.

arteriosclerotic alteration is focal in character, whereas senile changes show diffuse distribution." So the second group of changes is different from those of an arteriosclerotic nature and resembles more the normal senile alteration.

We have, then, a case showing the combination of arteriosclerosis and senile changes. Now the question is: Are these arteriosclerotic alterations with senile changes due to the myxedema, or are they independent of it? The patient was 66 years old, and it is quite possible to show arteriosclerotic alterations and even senile changes without any disturbance of the internal secretion. But one thing should be mentioned — the fatty degeneration of the ganglion cells is too great for the normal senile changes at this age, and that makes the question much more complicated.

It is, however, a common occurrence in senility for the thyroid to become atrophic and undergo diffuse sclerosis, destroying the gland tissue. Horsley declares that old age is only a form of mitigated hypothyroidism, and that the people who enjoy a green old age owe this happy condition to a thyroid which has remained normal. "The points of resemblance," says Crotti,[9] "between senility and a slight degree of hypothyroidism are more than one. Falling out of the hair, deafness, falling of the teeth, dryness of the skin, arteriosclerosis, bradycardia, diminution of the function of the nervous system and other symptoms which occur both in old people and hypothyroidism, can all be attributed to the atrophic condition of the thyroid gland." If this hypothesis is true, the question is very much simplified. For the increased arteriosclerosis and senile changes are nothing but the manifestation of the diminished function of the thyroid gland, and the pathologic findings which have been observed can safely be attributed to the hypothyroidism.

Schnitzler[10] described a case of a 32-year-old female, who had shown marked clinical symptoms of myxedema and mental symptoms of apathy and stupor and somnolence. He grouped his case with the type of which Spielmeyer wrote as "causes of senile dementia which differ from the usual type in that the marked dementia rapidly ensues, together with focal symptoms of asymbolic and aphasic character." He shows, however, as Fuller expressed it, an inclination to flirt with the rather fascinating idea of an origin from disordered internal secretions, since his case and one of the cases reported by Perusini exhibited certain myxedematous symptoms. "Although," Schnitzler says, "the thyroid gland could not be examined postmortem, since the patient became very much better under the administration of thyroid preparation, it is quite possible that we dealt with a thyrogeneous disease. So we have a case, showing a possibility of an intimate relationship between internal secretion and senile degeneration. If the normal

9. Grotti: Thyroid and Thymus, Philadelphia, Lea & Febiger, 1918.

10. Schnitzler: Zur Abgrenzung der sogenannten Alzheimerschen Krankheit, Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:34, 1911.

senile degeneration is merely a consequence of hypofunction of the thyroid gland, can this condition of senile changes in our case not be attributed to the complication of the myxedema?" It seems to me rather unnatural to have classed his case as senile dementia. I see no reason why it should not be grouped with the myxedematous psychoses. In his case he has found no senile plaques; he has observed Alzheimer's neurofibrillar change, but this is not at all pathognomonic of senile dementia or of other senile conditions. Goto[11] found typical Alzheimer's[12] change in feebleminded subjects, and even in dementia precox. I was unable to find any of these changes in any parts of the brain in this case.

Further, if we look over the mental symptoms, such as loss of memory, especially for recent events, disturbance of the attention, suspiciousness, acute dementia, etc., which are always found in senile cases as well as in this case, we cannot believe that the anatomic substrata of this disease is dependent on anything else than the senile changes in a broad sense — i. e., arteriosclerotic alterations, normal senile alterations and characteristic degeneration of senile dementia.

As for the cell change associated with the edematous condition, though it has, of course, nothing to do with senility, it must be considered as pathognomonic, since the edematous condition itself is the partial manifestation of the general symptoms of this disease.

The cerebellum was found more intensively degenerated than the cerebrum. The Purkinje cells diminished in number, those remaining showing marked degeneration. The fatty degeneration of the Purkinje cells, however, was not so marked as of the ganglion cells of the brain and the spinal cord which shows merely that the Purkinje cells are so-called lipophobic cells and nearly always escape fatty degeneration.

How far the cerebellar symptoms of reported cases and of the present case can be attributed to the pathologic anatomic conditions of the cerebellum is hard to answer, but it is quite probable that they have played more or less of a part in the existence of such marked symptoms.

SUMMARY

1. The writer presents a case of typical myxedematous psychosis which showed, together with known symptoms, marked disturbance of coordination, vertigo and somnolence.

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11. Goto: Alzheimersche Neurofibrillenveraenderungen in der Hirnrinde der Schwachsinnigen, Tokio Shinkeigakkai Zasshi, 1917.

12. Alzhemer: Die arteriosklerotische Atrophie des Gehirns, Allg. Ztschr. f. Psychiat. **51**:809, 1895. Beitrag zur pathologische Anatomie der Seelenstoerungen des Greisenalters, Neurol. Centralbl. **18**:95, 1899. Seelenstoerungen auf arteriosklerotischer Grundlage, Allg. Ztschr. f. Psychiat **59**:695, 1902.

2. The thyroid gland was found extremely atrophic with the gland tissue replaced by connective tissue fibers and with no isthmus. The remaining gland tissue was infiltrated by lymphatic cells. The writer suspects some etiologic relationship between the congenital factor and this disease.

3. The pituitary body was smaller than normal, though microscopically there was no evidence of atrophy.

4. The right ovary was removed ten years ago, the left remained atrophic, showing an evidence of chronic inflammatory process.

5. The writer suspects, having seen a similar condition of ovaries in a former case of the Danvers State Hospital series, some etiologic factors in certain diseased conditions of the ovaries.

6. The pathologic changes in the brain and the cerebellum consist of arteriosclerotic alterations, general senile changes and cell change associated with the edematous condition.

7. The writer attributes the arteriosclerotic alterations and the general senile changes to the effect of the myxedematous disease.

8. The cell change associated with the edematous condition is considered as pathognomonic.

The writer believes also that there is a possible correlation between the marked atrophy of the cerebellum and the clinical symptoms of disturbed coordination and of vertigo.

The writer is indebted to the staff of the Danvers State Hospital, especially to Drs. Macdonald, Bryan and Stevenson, for completing this report. He is also indebted to Drs. Southard and Canavan for their encouragement and advice in his study. To all of these he wishes to express his appreciation.

In addition to the references already given, the following will be found of interest:

Barham, G. F.: Insanity with Myxedema, J. Ment. Sc. **58**:226, 1912.

Alzheimer: Ueber eine eigenartige Erkrankung der Hirnrinde, Allg. Ztschr. f. Psychiat. **64**:146, 1907.

SENSORY CHANGES IN INJURIES OF THE MUSCULOSPIRAL NERVE *

ARTHUR S. HAMILTON, M.D.

MINNEAPOLIS

In view of the several very brilliant and equally exhaustive reports on the innervation of the skin which have appeared in the literature of the last few years, one must, with some temerity, approach the study of the sensory functions of any one of the peripheral nerves. And this is the more true when one considers that much of the work recently published has been done on patients well fitted both by education and by training to carefully analyze their sensations and by workers who have given to the task many months of painstaking examination, whereas the conditions obtaining in army life render a similar study wholly impossible, partly by lack of that preliminary education and training on the part of the patient which is essential to the best results in interpreting new and confusing sensations, and partly by reason of the lack of time which permits only a limited study in a routine examination. The difficulty which the latter factor offers is appreciated when one recalls the statement of Trotter and Davis[1] that it requires from forty-five to fifty hours to examine and record the sensibility to cold alone in a fairly large area. For these reasons, therefore, no attempt has been made to describe adequately and to measure all the different sensory disturbances that may appear in the region supplied by the musculospiral nerve. The excuse for this report must lie in the scarcity of literature dealing with the sensory field of this particular nerve and in its surprising areas of distribution found in numerous cases of nerve wounds among soldiers — surprising at least in view of the anatomic distribution of the nerve as commonly taught.

It is important to mention that, as all the cases here reported were studied at the Walter Reed General Hospital in Washington, D. C., it was not possible to determine the area of sensory change in the early stages of the injury and in several instances well marked evidences of healing had appeared before the study was begun. It was especially in those cases in which the loss of function of the musculospiral nerve was dependent on fracture of the humerus, with the development of scar tissue and which presented a cessation of function but not necessarily an anatomic loss of continuity, that the picture was at times

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* Read before the meeting of the American Neurological Association, held in Atlantic City, N. J., June 16-18, 1919.

1. Trotter and Davis, Brain **38**:137 (Feb. 9) 1919.

greatly altered, for in from three to five or six months that had usually intervened before the study was made, marked improvement could, and often had occurred.

In many cases the amount of destruction of tissue, in addition to the actual section of the nerve, has seriously interfered with the work, often by the injury or section of nerves, especially of the musculocutaneous, in addition to the one desired to be studied, thereby confusing the sensory picture. So far as possible, however, cases were chosen for special study in which the section of the nerve appeared to have been at least physiologically complete, in which a sufficient period of time for healing to occur had not intervened, in which the wound was small and appeared not to have involved nerves other than the musculospiral and in which the intelligence of the patient was such as to permit a reasonably reliable study. It must be admitted, however, that when all these restrictions were applied, the number of cases suitable for study was so much reduced that the limits were more than once overstepped.

The total number of musculospiral cases studied was fifty-five, and in all of these the investigation was carried far enough to determine the area of sensibility to pressure, pin prick and cotton or camel's hair touch, but beyond these routine tests, the type of study made varied so much and in many of the cases is still so incomplete that no absolute percentages of the difference findings can be given.

THE SENSORY BRANCHES OF THE MUSCULOSPIRAL NERVE

There are three sensory branches of the musculospiral nerve, distributed in the average case as follows:

The internal cutaneous branch is given off in the axillary fossa and supplies the internal part of the posterior surface of the upper arm below the deltoid and as far down as the point of the elbow. On account of its high origin, this branch is affected in only a small number of cases, in which the musculospiral nerve is injured at a very high point. The external cutaneous branch is given off at a point where the musculospiral nerve passes around the external edge of the humerous and supplies the external part of the posterior surface of the arm, the lower part of the outer surface of the arm and a more or less narrow band of the posterior surface of the forearm lying between the musculocutaneous and the internal cutaneous nerves. Often there are two external cutaneous branches, and in such cases the area of numbness will be different if only one branch is involved. The superficial radial nerve has its origin at, or just below, the elbow joint, and after passing down the forearm is distributed to the skin of the external part of the dorsum of the hand, to a variable but

usually small part of the thenar eminence, to the dorsal surface of the thumb, to the dorsal surface of the first phalanx of the index finger and to the outer half of the dorsal surface of the first phalanx of the second finger (Fig. 1).

With so wide an anatomic distribution and with its well known extensive motor supply, one might expect a very widespread and complete loss of sensation in the various injuries to which the musculospiral nerve is subjected. As a matter of fact, quite the contrary is true, and the area of sensory loss is sometimes surprisingly small and is almost always incomplete.

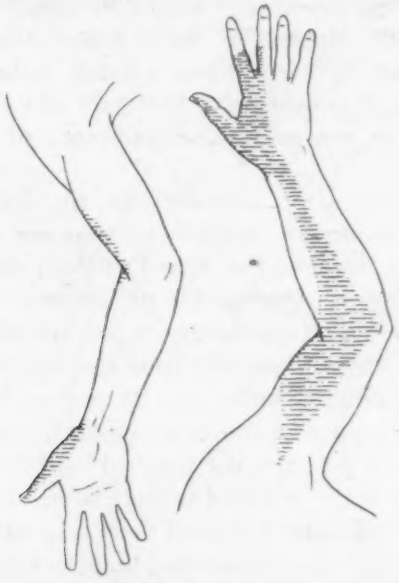

Fig. 1.—Area of sensory distribution of the musculospiral nerve as given by Tinel in Nerve Wounds, p. 103.

One of the unexpected findings in all nerve injuries is the failure of the individual to recognize subjectively and with any degree of accuracy the area of sensory loss, especially of epicritic loss and particularly so in the area of distribution of the musculospiral nerve. Frequently patients with well marked paralysis of the extensors of the hand, following injury, if asked for their subjective sensory area will say they have none. If their attention is insistently brought to the matter, they will tell of a small area of numbness over the first interosseous space and over the dorsum of the thumb and perhaps over the dorsum of the first phalanx of the index finger, and they will be greatly surprised when a larger area of sensory loss can be demonstrated.

As already explained, the internal cutaneous branch rises so high that it is seldom involved in injury of the musculospiral nerve and

even the external cutaneous frequently rises above the area of injury, at least its supposed area is often found not involved even in injuries high up in the arm. It is impossible, however, to explain all the variations found in the area of numbness on the basis of the point of origin of the sensory branches, and other explanations are offered, chiefly that of the overlapping or anastomosis of the musculocutaneous, internal cutaneous, and median nerves involving the musculospiral field and giving varying areas of sensory loss when these nerves are injured. In the upper arm the intercostohumeral or the lesser internal cutaneous may supply areas ordinarily innervated by the internal cutaneous branch of the musculospiral and may also supply that part of the upper arm ordinarily supplied by the external cutaneous branch. I have also recently seen a case in which a lesion of the circumflex nerve caused a large area of numbness in that part of the upper arm ordinarily supplied by the external cutaneous branch of the musculospiral nerve.

As is so common after severance of any large nerve of the arm, the patient will usually complain of complete loss of feeling in all parts of the arm distal to the wound, directly following injury to the musculospiral nerve. During this preliminary period the arm is described as a very heavy appendage, or the statement is made that the arm feels as if wholly separated from the body and must be seen or felt by the other hand in order that the patient may appreciate its presence. After the lapse of a few hours or days, the patient discovers that there is feeling in the parts not supplied by the nerve immediately injured and the area really involved is more or less clearly recognized.

In the fifty-five cases that I studied there was only one instance in which the musculospiral nerve alone had been severed sufficiently high to produce a loss of sensation corresponding to all three branches.

REPORT OF CASES

CASE 1.—The previous history was not noteworthy. October 13, 1918, the patient while holding his left arm in the position of guard with the forearm horizontal, was struck below the clavicle by a shell fragment which punctured the lung and passed out the back. His left arm immediately became numb and fell to his side. There was no jerking or twitching. He felt as if the arm were shot off. His neck was stiff and he was unable to look down. He repeatedly asked those about him if his arm were not really gone. For some time after the injury he would seem to feel his arm in the position of guard and would reach up with his other arm to scratch it. At first he had excessive pain in the shoulder; later his whole arm ached a good deal, but he could not localize the ache as any worse in one part than in another. There was a slight causalgia for a time in the median. He was told that his musculospiral nerve was sutured directly after his injury, but the record is not clear on this point. At the examination, in February, 1919, he was conscious only of some numbness on the dorsum of the index finger, which he thought worse

in the first phalanx than in the second, and of some numbness on the back of the upper arm. An examination made with considerable difficulty, at this time, showed sensory changes as seen in Figure 2. Since February there has been some improvement in the sensory and motor condition and on May 31 even epicritic sensibility had returned over the dorsum of the hand with the exception of the area over the first metacarpal bone. The analgesic islands, outlined in the diagram, persisted. The dorsum of the first phalanx of the index finger was anesthetic and hypalgesic. Tinel's sign was elicited as far as the elbow and at this point stroking produced a tingling feeling in the fingers.

In a general way this case conforms fairly well to the typical distribution of the musculospiral nerve. The fact that sensibility is more disturbed in the upper part of the forearm than at the wrist was a

Fig. 2 (Case 1).—Area of sensory disturbance. In this and in the accompanying illustrations the meaning of the legends employed is given as a part of each illustration. When lines lie close together it is not intended that they should represent the relative limitations of the different sensory fields. They were not permitted to overlap merely as a matter of clearness of representation. In most cases also the work was not sufficiently carefully done to permit the accurate tracing of the relative sizes of the different fields.

frequent finding in our cases. The enlargement of the sensory area at the wrist and just above is atypical, though found in one other case, and on stroking touch the patient was able to identify an area of decreased sensibility even to the extreme ulnar side of the arm, just above the wrist joint.

In the fifty-five cases there were twenty-seven with a definite area of sensory involvement in the forearm as well as the hand, and one

instance of involvement of both musculospirals, each case involving the forearm. Seven of these were rejected on account of various complications. The subjective sensory phenomena were usually about as in Case 1, with an initial sensation much as if a heavy blow had struck the arm and a subsequent feeling of loss of the limb and a still later more clearly defined and localized loss of sensibility. The following is a fairly typical case except that there seems to have been less initial loss of sensibility than was ordinarily present.

Case 2.—While standing the patient was struck by a rifle bullet in the upper portion of the left upper arm, Sept. 15, 1918. The arm was in a horizontal position, and he had the sensation of being struck a heavy blow. The arm rotated inward, bringing the palm downward. The fingers flexed like a claw

Fig. 3 (Case 2).—Area of sensory disturbance.

and remained so for some time. There was no pain except at the first blow and no feeling of the arm being gone. He did not recall noticing much numbness at any time. His record states that he was operated on the night of the 15th and his musculospiral nerve sutured.

When examined, May 7, 1919, a rather extensive area of sensory disturbance was found (Fig. 3). There was no connection between the area on the forearm, which extended well over into the supposed region of the musculocutaneous nerve, as did some of the others, and the area on the hand. There was a loss of sensibility to cold at 2 C. slightly wider than the area of touch loss on the forearm and especially extending considerably farther over on the palmar surface of the forearm than any other form of loss of sensibility. Two point discrimination was greatly decreased on the dorsum of the forearm (33 per cent. of correct answers at 110 mm. with 53 per cent. of correct answers at 50 mm. on the right forearm). On the dorsum of the hand two point sensibility could not be measured on account of the inability to perceive pressure.

Joint sensibility was normal in the fingers and thumb. Vibration sense was normal in the fingers and somewhat reduced over the thumb.

Case 3.—While kneeling and digging in, the patient was struck by shrapnel in two places on the left upper arm and in six or seven places on the right upper arm, Oct. 5, 1918. He was also struck in the right knee and thigh. He fell and was unconscious at least a few minutes. On recovering consciousness, both hands and arms were wholly paralyzed for motion and sensation and remained so for one day. The left hand and forearm felt numb on the dorsum until the end of January and then began rapidly to improve. There was some numbness in the musculospiral area of the right hand and lower part of the forearm for two weeks with rapid improvement following.

When examined, May 9, 1919, there was a slight hypesthesia and hypalgesia in the right hand with a subjective area practically the same in extent (Fig. 4).

Fig. 4 (Case 3).—Area of sensory disturbance.

May 28, 1919, it was impossible to map any definite area of disturbed sensibility in the right hand or arm.

May 9, 1919, there was an extensive area (Fig. 5) of diminished touch and pain sensibility on the posterior surface of the left hand and arm, most marked on the forearm. May 28, 1919, the area was considerably diminished and could be outlined in anything like its original form only by stroking touch. There was no loss of appreciation of weight but two point discrimination was definitely lessened on the dorsum of the forearm and a small area of somewhat lessened appreciation of cold at 4 C. was found on the dorsum of the forearm a little below the elbow.

Case 4.—The patient while advancing was struck by shrapnel in the left upper arm and left leg, Nov. 1, 1918. His arm felt dead but he could move it at the elbow directly after receiving the wound. He could not move the fingers; the arm did not jerk or twitch. The next day he noticed a definite numb area on the back of his left hand, forearm and arm. He could move the arm at the shoulder but not at the elbow or wrist. The fingers could be feebly flexed.

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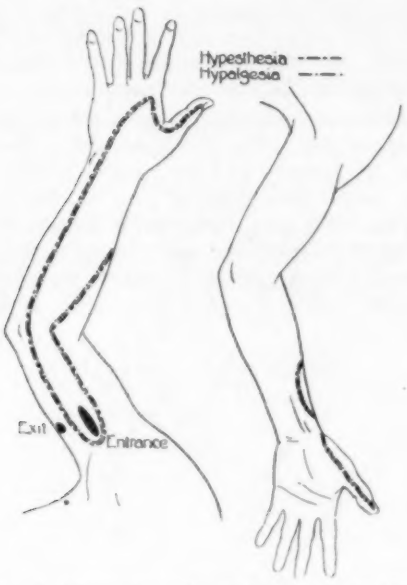


Fig. 5 (Case 3).—Condition, May 9, 1919.

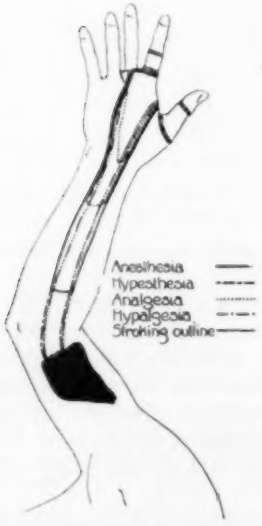


Fig. 6 (Case 4).—Area of sensory disturbance.

When examined May 13, 1919, he had a large scar, 6 by 2½ inches, over the lower end of the outer side of the left upper arm, adherent to the bone. The area of sensory disturbance (Fig. 6) extended down the back of the forearm in a rather narrow line and in the hand there was loss or great diminution of touch and pain over the first phalanx of the thumb but the skin over the first metacarpal and over the first interosseous space responded normally. In this case the loss of sensibility was more complete in the areas affected in the hand than in the forearm and in the lower part of the forearm than in the upper.

A further examination, May 28, 1919, showed an area of complete loss of sensibility to temperature of 5 C. and to ice over the region of greatest sensory loss on the hand, but on the thumb similar temperatures were fully appreciated. Joint sensibility was normal. Pressure sense was greatly diminished on the hand (200 gm.) and much less diminished on the forearm (27 gm.). On the hand, in two point discrimination, he gave 47 per cent. of correct answers at

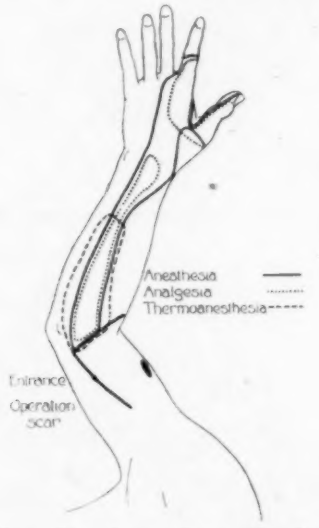


Fig. 7 (Case 5).—Area of sensory disturbance.

40 mm. as against 53 per cent. at 22½ mm. on the right hand. On the left forearm he gave 53 per cent. of correct answers at 80 mm. and on the right forearm 53 per cent. of correct answers at 47 mm.

In July he went to the beach and both forearms became much sunburnt. The left was a little more affected than the right and almost blistered, but the two felt equally sore and hot.

CASE 5.—Sept. 27, 1918, while walking, the patient was struck by a machine-gun bullet in the right leg and by a high explosive fragment in the left upper arm. The humerus was fractured. The left hand dropped immediately at the wrist and there was subsequently no movement at the wrist joint, though he could flex the fingers slightly. The hand and arm from the elbow down were numb on the dorsal surface, but he retained the sense of feeling on the palmar side and there was no pain except at the site of injury.

When examined, May 28, 1919, the ordinary line of numbness (Fig. 7) was found on the posterior surface of the forearm and thumb and the first and part of the second phalanges of the first finger were involved, but the skin

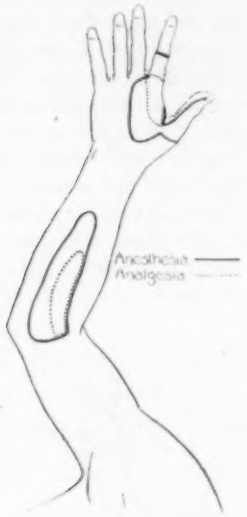


Fig. 8 (Case 5).—Condition, June 30, 1919.

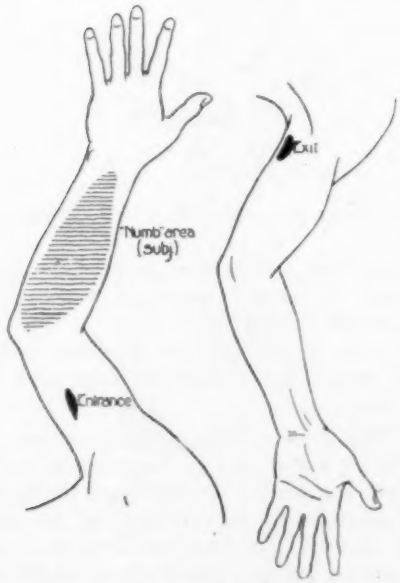


Fig. 9 (Case 6).—Area of subjective numbness.

over the first metacarpal and a part of the first interosseous space was unaffected. Pressure sense was much more affected in the hand and wrist than in the middle and upper part of the forearm, whereas two point discrimination was much more deficient in the upper part of the left forearm than in the wrist or hand. The area of loss to cold was wider than that of loss of touch and pain. There was no diminution of vibration or joint sensibility.

On June 30, 1919, the loss of sensation was about the same on the dorsum of the hand and upper two thirds of the forearm, but had quite disappeared over the wrist and lower one third of the forearm (Fig. 8).

Case 6.—Oct. 14, 1918, the patient, while standing, was struck by a fragment from a shell which exploded about 3 feet from him. He sustained a severe compound, comminuted fracture of the left humerus. He bled considerably and was unconscious for three or four days. His arm was heavily bandaged when he regained consciousness. His idea of the sensory loss was not exact,

Fig. 10 (Case 6).—Area of sensory disturbance.

but he was sure that at least he had had numbness of the back of the left thumb, of the proximal part of the left index finger and did have feeling somewhere toward the ulnar side of the hand. There was complete wrist drop.

When examined, May 5, 1919, the area of subjective numbness (Fig. 9) included a large portion of the dorsum of the left forearm, but he was not aware of any loss over the hand. The examination showed (Fig. 10) a definite area of anesthesia and analgesia of the dorsum of the thumb and of the wrist and anesthesia of practically the entire dorsal region of the forearm and of the lower part of the upper arm with a less wide area of analgesia. On the volar surface of the forearm, toward the radial side, there was hypesthesia and over the outer side of the thenar eminence and thumb there was anesthesia and analgesia. There were small areas of hyperalgesia over the distal phalanx of the thumb and over the knuckle of the index finger.

The involvement of the radial side of the forearm, in this case, suggests the possibility that the musculocutaneous was injured, but it may at least be said

Fig. 11 (Case 6).—Condition, June 1, 1919.

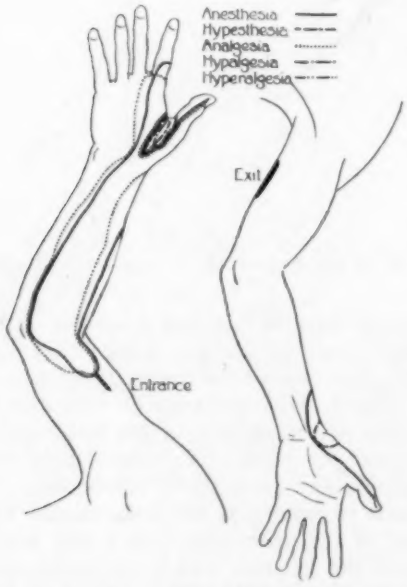


Fig. 12 (Case 7).—Area of sensory disturbance.

that the injury was not in such a position as to be likely to involve the musculocutaneous, unless it did so above its supply to the biceps and there was no evidence of any motor involvement to the biceps.

Examination, June 1, 1919, showed (Fig. 11) hyperesthesia and hyperalgesia and loss of cold at 5 C. over the area of the hand previously showing sensory loss, no evidence of any sensory involvement of the dorsum of the wrist and a decided diminution of the area on the forearm.

CASE 7.—The patient was struck by a machine-gun bullet, Sept. 29, 1918, causing compound, comminuted fracture of the left humerus. Following the injury there was extreme pain in the whole arm and considerable hemorrhage. He does not recall whether there was loss of feeling or motion following the injury. He was operated on thirty-six hours later, and is sure there was no paralysis of motion following the operation. The arm was placed in a Thomas splint with extension. Three weeks later he noticed that wrist drop was

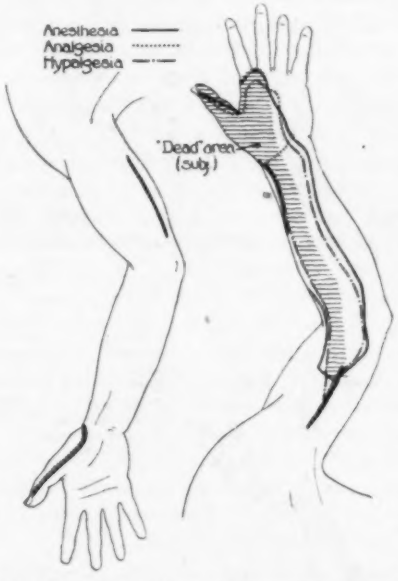


Fig. 13 (Case 8).—Area of sensory disturbance.

developing and that the end of the thumb was growing numb. The loss of motion and sensation increased for a time, but he thinks that during the past few months there has been progressive improvement.

When examined, Aug. 19, 1919, a wide area of sensory change was found (Fig. 12). Sensibility was much less disturbed in a narrow line over the first interosseous space than in the areas to either side.

CASE 8.—Sept. 30, 1918, while standing, the patient was struck on the outer side of the right upper arm and the bullet came out on the inner side. Directly following the injury, he had no power of sensation or motion in the right arm or hand. Seven hours after the injury he was operated on and the musculospiral nerve sutured. Just before the operation, he found he could flex the right fingers feebly. After the operation there was numbness down the outer side of the right arm, on the dorsum of the forearm and on the radial side of the hand.

Fig. 14 (Case 9).—Area of sensory disturbance.

Fig. 15 (Case 10).—Area of sensory disturbance.

April 8, 1919, examination showed (Fig. 13) an extensive loss of sensation on the dorsum of the hand and forearm and on the outer side of the upper arm. Diminution of pain and touch was much more marked distally than proximally in the forearm and more marked in the hand than in the forearm or arm. Vibration and joint sensibility were normal but two point discrimination was diminished. The mental condition of the patient did not permit a very satisfactory examination.

Case 9.—Sept. 29, 1918, the patient was struck by a piece of shrapnel on the dorsal surface of the right arm just below the elbow. The fragment passed out through the ulnar surface. Directly after the injury was received the entire extremity from the elbow down felt numb and "tingled as if a nerve had been struck." He was operated on the following day, but thinks no nerve was sutured. Subsequently he found about the same area of numbness on the dorsum of the arm and hand as at present, except that it was slightly larger. At an examination made May 26, 1919, it was found that the loss of sensibility (Fig. 14) was much more complete on the dorsum of the forearm than on the hand, and there was an area of normal sensibility over the first metacarpal and the first interosseous space. There was no perceptible loss of sensation on the index finger, but the dorsum of the first phalanx of the second finger showed hypesthesia. Vibration sense was practically normal.

Case 10.—While lying down, Oct. 24, 1918, the patient was struck on the outside of the left upper arm by a machine-gun bullet. He felt "as if a hot poker were being run through it and the arm felt numb from the wound down." When dressed he could feel the bandages about the palm of the hand and the ulnar part of the wrist but not anywhere on the forearm. Feeling began to return in the forearm about the middle of November. When examined, June 4, 1919, there was a large area on the dorsum of the forearm (Fig. 15) of very marked sensory disturbance. Anesthesia was wider than analgesia. The margin of stroking outline lay outside both and the line of thermoanesthesia and of thermohypesthesia (8 C.) was still wider. The dorsum of the thumb showed anesthesia and analgesia, but the dorsum of the first metacarpal and of the first interosseous space showed hypesthesia and, over part of the former, hypalgesia. Vibration sense was reduced on the thumb. In two point discrimination he gave 53 per cent. of correct answers at 25 mm. in the right forearm and 53 per cent. of correct answers at 70 mm. in the left forearm.

Case 11.—The patient was struck in the left upper arm Nov. 7, 1918, by a machine-gun bullet which caused a compound, comminuted fracture at the middle of the humerus. There was immediate and complete motor paralysis of the musculospiral nerve, but he is uncertain as to the sensory condition. Examination, April 24, 1919, revealed anesthesia and analgesia (Fig. 16) of the area on the second metacarpal and the second interspace and hypesthesia and hypalgesia on the dorsum of the thumb and the first metacarpal with a narrow band of normal sensibility between them. There was also hypesthesia and hypalgesia over a band of considerable width extending down the dorsum of the forearm. June 21, 1919, the area on the forearm was considerably reduced. The condition of the hand was not much altered except for hyperalgesia over the thumb and an increase in the size of the normal band over the first interosseous space. Aug. 16, 1919, no sensory loss could be discovered in the forearm, and the hand was greatly improved (Fig. 17).

Case 12.—While the patient was asleep, Oct. 15, 1919, a bullet passed through the left forearm, just below the elbow. Directly the entire extremity was numb from the elbow down. Feeling later returned in all parts except in a line down

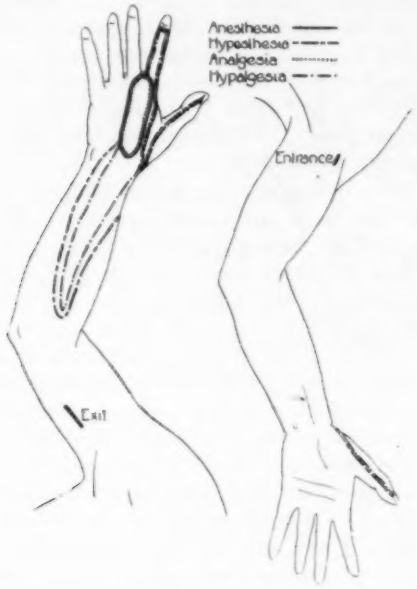


Fig. 16 (Case 11).—Area of sensory disturbance.

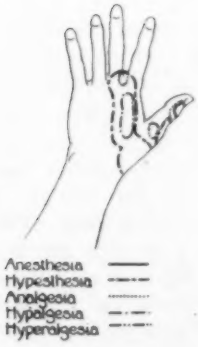


Figure 17

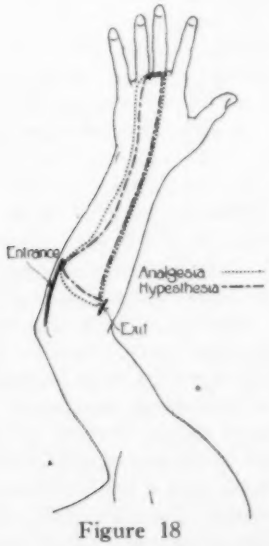


Figure 18

Fig. 17 (Case 11).—Area of sensory disturbance, Aug. 16, 1919.
Fig. 18 (Case 12).—Area of sensory disturbance.

the center of the back of the hand and forearm. Examination, July 10, 1919, showed hypesthesia and analgesia in the area shown in Figure 18 and a partial paralysis of the musculospiral motor supply.

CASE 13.—Nov. 1, 1918, the patient was struck on the inside of the left upper arm by a bullet which was removed on the upper side of the upper arm. So far as the patient knows, there was no motor or sensory loss following this operation, but about March 1, 1919, a sequestrum was removed from the upper arm and subsequently he had difficulty in extending the fingers and thumb. When examined, June 25, 1919, there was still no extension of the thumb or fingers of the hand, but extension of the wrist was fair. On the dorsum of the forearm was an area 1¾ by 1½ inches (Fig. 19) of loss of sense of touch. In this area pin prick was described as "less sharp," but as "hurting more," with a feeling of being different from a normal pin prick. No other sensory disturbance could be found.

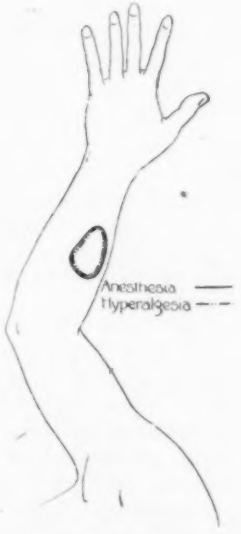


Fig. 19 (Case 13).—Area of sensory disturbance.

There were twenty-seven cases in which the sensory phenomena, due to injury of the musculospiral nerve, were limited to the hand or to a region extending slightly above the wrist. Of these, fourteen were selected for reporting.

CASE 14.—While riding a motorcycle, Nov. 9, 1918, the patient's left arm dropped suddenly to his side. He was not conscious of having been injured, and there was no pain or jerking of the hand or arm. An examination showed he had been struck in the left arm by a bullet. He could flex his fingers, and he is sure that the dorsum of the thumb and of the first finger and the space between were numb and that he could not appreciate pin prick in this region as pain.

When examined, April 17, 1919, only an area on the dorsum of the first phalanx of the thumb and a small area on the outer surface of the dorsum of the first phalanx of the index finger felt numb. Examination showed (Fig. 20) analgesia to 12 gm. of pressure over the knuckle of the index finger and to

Fig. 20 (Case 14).—Area of sensory disturbance.

Fig. 21 (Case 15).—Area of sensory disturbance.

10 gm. of pressure over a band extending back about half way to the wrist. The dorsum of the first and second phalanges of the thumb was analgesic to 12 gm. of pressure. Both areas were anesthetic to touch as was also an area extending proximally from the knuckle almost to the wrist. He gave two point discrimination in 53 per cent. of tests over the normal hand at 21¼ mm. and in 53 per cent. of tests over the affected area at 47 mm. On the area over the knuckle, he could barely discriminate 25 gm. of pressure. With the exception of this region, discrimination of pressure was not greatly reduced in the affected area.

CASE 15.—The patient was injured Oct. 3, 1918, by a machine-gun bullet entering the right arm at the elbow. He was sitting with his elbow on his knee. He felt as if a "log had struck him" and was knocked over but did not become unconscious. There was no twitching or jerking in the hand or arm. The dorsum of the thumb and of the back of the hand (with the exception of the extreme ulnar side) was numb. He cannot recall anything concerning the feeling in the back of the fingers, but does not remember any numbness, and he is positive there was no numbness in the forearm. There was some stinging feeling in the back of the hand for a time, but this had disappeared at the examination on April 18, 1919, when he showed an area of anesthesia and analgesia as outlined in the diagram (Fig. 21).

A second examination on June 10, 1919, showed some improvement in sensation. Vibration and joint sensibility at this time were normal. Cold at 5 C. was readily felt. In discrimination of two points he gave 53 per cent. of correct answers at 45 mm. in the right hand and 100 per cent. at 26 mm. in the left hand.

CASE 16.—Aug. 10, 1918, while lying flat on his face, the patient was struck by a fragment of shell which entered the left arm 2 inches below the elbow on the radial side and passed out through the posterior surface of the arm just above the elbow, fracturing the humerus. Directly after he was struck, the hand and arm drew up and the "fingers clenched," after which the entire arm became limp and numb. He felt as though the arm from the elbow down did not belong to him, and he could make no movement from the hand or forearm.

When examined, May 23, 1919, there was an area (Fig. 22) of anesthesia and hyperalgesia over the phalanges of the thumb and of anesthesia and analgesia over the second metacarpal bone and second interspace and over most of the first interspace. Over the first metacarpal bone there were hypesthesia and hypalgesia. Sensibility to cold at 2 C. was lost in all this area and over a considerable area over the thenar eminence. Cold at 22 C. was lost over a somewhat wider area. The threshold for two point discrimination was about one-half higher in the affected arm than on the unaffected side. Weight discrimination was very little changed. In the first, second and third fingers there was an area of uncertain loss to touch, probably due to local skin condition.

CASE 17.—July 16, 1918, a fragment of shell entered over the lower end of the left biceps and passed out a little above the outer condyle of the humerus. When examined, May 26, 1919, there was anesthesia (Fig. 23) over the entire dorsum of the thumb and over the radial side of the dorsum of the hand with the exception of a band about 1 inch wide extending down the first interspace. Pin prick was appreciated as such at from 4 to 8 gm. of pressure; two point and weight discrimination were both only slightly impaired in the same region. There was no loss of joint or vibration sensibility. An area of hyperalgesia was found over the web of the thumb.

Fig. 22 (Case 16).—Area of sensory disturbance.

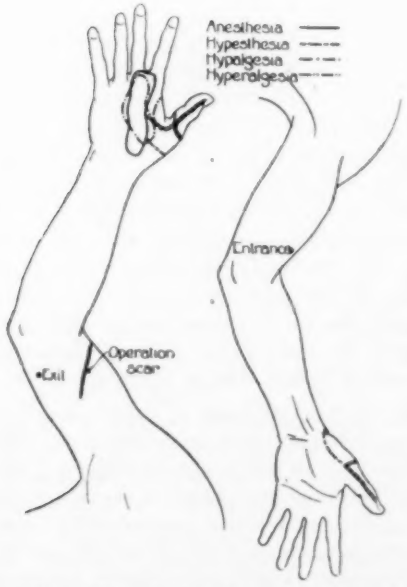


Fig. 23 (Case 17).—Area of sensory disturbance.

Case 18.—The patient was injured in the left arm, on the outer side, just above the elbow by a piece of shrapnel, Oct. 4, 1918. He was knocked down, and remained unconscious for a few minutes. On recovering, he felt as though the arm had been blown off. When he attempted to move it, it hung limply by his side, and he said it "felt as if it weighed a ton." The next morning there was numbness in the area shown in the diagram and over the dorsum of the first finger. He is positive there was no numbness in the arm. He noticed improvement first in the distal part of the forefinger and then in the region of the first metacarpal.

Fig. 24 (Case 18).—Area of sensory disturbance.

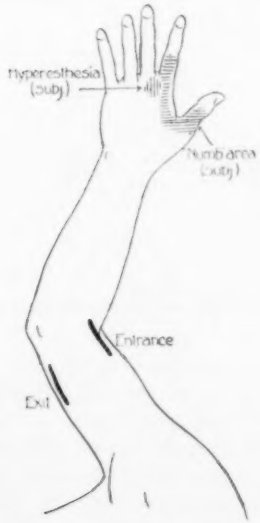


Fig. 25 (Case 19).—Area of sensory disturbance.

When examined, May 12, 1919, a roentgenogram showed some small fragments of bone still in the wound and in the region of the musculospiral nerve. There was an area of anesthesia to touch and analgesia to prick over the second and third metacarpals as shown in Figure 24 and outside of this there was an area of hypesthesia and hypalgesia. The dorsum of the phalanges of the thumb and of the distal part of the first metacarpal was anesthetic and analgesic. In the first interosseous space was an area of normal sensibility.

Case 19.—Oct. 29, 1918, the patient was struck in the left arm just to the outer side of the elbow. The missile passed out at a slightly higher level on the posterior surface of the upper arm, fracturing the humerus. His arm was immediately paralyzed from the elbow down and subsequently there was an area of "numbness" (Fig. 25) involving the dorsum of the first phalanx of the thumb, the distal part of the first interosseous space, a band over the second metacarpal and the first phalanx of the forefinger. Over and just proximal to the knuckle of the second finger was a circular area of subjective hyperesthesia. Examination, May 16, 1919, showed (Fig. 26) hypesthesia and hypalgesia over the first phalanx of the index finger, analgesia and hypesthesia over the thumb and anesthesia and analgesia over the remainder of the area. The subjective hyperesthetic area gave normal response to touch and prick.

Fig. 26 (Case 19).—Condition, May 16, 1919.

Case 20.—The patient was injured Oct. 4, 1918, by a machine-gun bullet. He was lying down, and the bullet entered at the lower part of the right deltoid on the outer surface, and came out on the inside of the arm just below the axilla. The whole arm felt numb subsequently, and motor power was completely lost. After a few hours the arm was less numb and began to feel "sore" but not in any special locality. At the same time the numbness largely disappeared from the upper extremity but for three weeks there was some loss of feeling in the forearm, not clearly localized. There has been no numbness since. After this there was not much change in feeling but marked improvement in motion.

When examined, May 13, 1919, he outlined an area of numbness on the back of the hand a little smaller than that found on examination and an area on the inside of the hand considerably greater than could actually be demonstrated. Examination showed (Fig. 27) anesthesia and analgesia over the dorsum of the phalanges of the thumb, over the first and part of the second

Fig. 27 (Case 20).—Area of sensory disturbance.

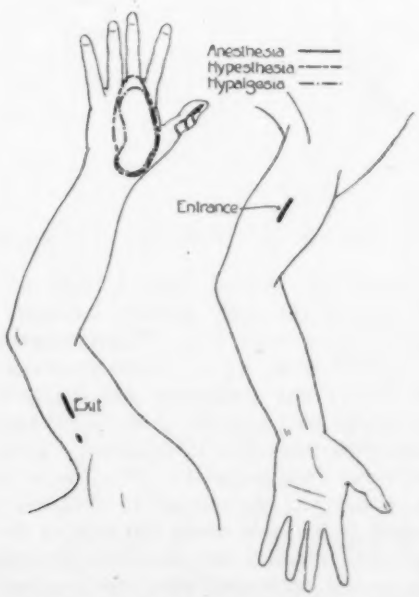


Fig. 28 (Case 21).—Area of sensory disturbance.

metacarpals, including the first interosseous space, with an area of hypesthesia and hypalgesia considerably wider. There was also hypalgesia over the first and second phalanges of the right index finger.

CASE 21.—The patient was hit by a machine-gun bullet, Oct. 16, 1918, in the upper part of the left arm, and the humerus was fractured. He felt no pain and did not know that he was hurt, until his arm, on which he was leaning, gave way. For one week the arm felt numb up to the shoulder and after three weeks there was no sensory loss of which the patient was aware other than the area shown on examination, June 5, 1919 (Fig. 28). The area of hypesthesia on the dorsum of the hand was wider than the area of hypalgesia. The dorsum of the second phalanx of the thumb was anesthetic and hypalgesic. The dorsum of the distal half of the first phalanx was anesthetic and of the proximal half hypesthetic, but he could feel pin prick in both places.

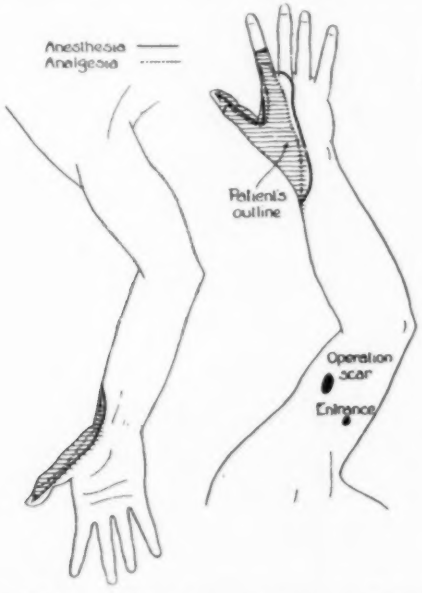


Fig. 29 (Case 22).—Area of sensory disturbance.

CASE 22.—The patient was injured, Nov. 1, 1918, in the right arm and forearm. The only part of the early history available, was that after the injury was received he had a wrist drop. When examined, May 2, 1919, he had slight extension of the wrist, a very slight extension of the fingers and none of the thumb. There was anesthesia and analgesia (Fig. 29) of the dorsum of the thumb, of the first phalanx of the index finger and of the radial side of the hand, extending upward a little distance above the wrist. Anesthesia was somewhat wider than analgesia. The area of stroking change was slightly larger than either and the subjective area was somewhat smaller. Sensation was preserved in a region along the edge of the web of the thumb.

June 9, 1919 (Fig. 30), sensation had improved decidedly. The dorsum of the thumb and of the second metacarpal remained anesthetic and analgesic but the area between was very slightly hypalgesic. Loss of sensation of cold at 8 C. and the stroking outline almost coincided. On the front of the right wrist was a region of increased sensibility to cold at 8 C.

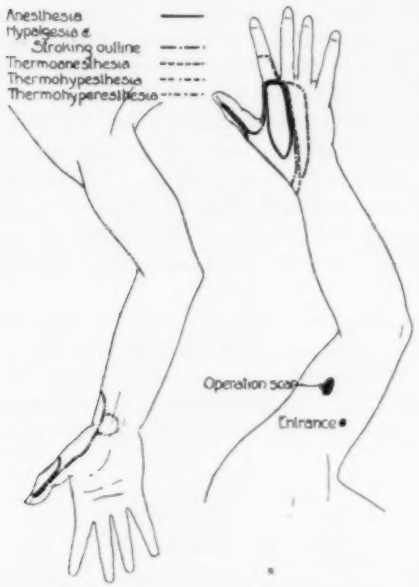


Fig. 30 (Case 22).—Improvement in sensation, June 9, 1919.

Figure 31

Figure 32

Fig. 31 (Case 23).—Area of sensory disturbance.
Fig. 32 (Case 24).—Area of sensory disturbance.

Case 23.—The patient was struck by shrapnel, Oct. 9, 1918, in the lower one-third of the right upper arm, receiving a compound, comminuted fracture of the humerus with considerable loss of bone. When examined, May 16, 1919, there was considerable loss of substance of the shaft of the humerus, a complete motor paralysis of the musculospiral supply and an area of anesthesia and analgesia on the dorsum of the hand (Fig. 31).

Case 24.—Oct. 14, 1918, a bullet passed through the right arm, just below the elbow, resulting in musculospiral paralysis. The history of the early symptoms is lacking. When examined, May 22, 1919, there was (Fig. 32) hypesthesia and hypalgesia of the dorsum of the thumb and of the radial side of the hand. Extension at the wrist was poor and of the thumb and fingers was entirely lacking.

Case 25.—The patient was shot through the left elbow, Oct. 12, 1918. Musculospiral paralysis resulted. The early history was not available. When examined, June 30, 1919, there was fair extension of the wrist, fingers and thumb, but there was a large area of anesthesia and analgesia on the dorsum of the hand and an area of hypesthesia and hypalgesia on the dorsum of the thumb (Fig. 33).

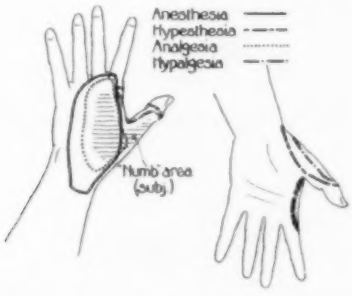


Fig. 33 (Case 25).—Area of sensory disturbance.

Case 26.—The patient was struck on the outside of the right arm just above the elbow, Oct. 8, 1918, by a machine-gun bullet which came out on the posterior and inner surface at about the same level. Motion was lost at once in the musculospiral supply and for a time the arm felt numb, but he thinks he could distinguish touch at all times. He recognized no complete loss of sensation. When examined, June 5, 1919, there was a small area of anesthesia (Fig. 34) about the knuckle of the right index finger and on the dorsum of the second phalanx of the thumb with hyperalgesia in both areas and inability to extend the thumb. There was also rather weak extension of the fingers.

Case 27.—The patient was injured Oct. 8, 1918, by high explosive fragments which entered the left upper arm and forearm. The arm at once became limp and he felt as though he had lost it. There was no sensation at any point on the arm until the following day, after which sensation returned except in the area shown in the diagram. When examined, April 24, 1919 (Fig. 35), the entire dorsum of the thumb was anesthetic and there was a small area of analgesia on the distal part of the dorsum of the first phalanx.

Case 28.—Oct. 15, 1918, the patient was struck by high explosive fragments at various points, including the front of the right axillary fold. The entire right arm and hand were paralyzed for motion and sensation for some hours

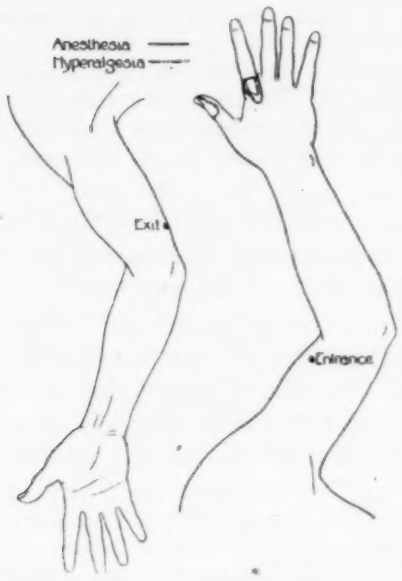


Fig. 34 (Case 26).—Area of sensory disturbance.

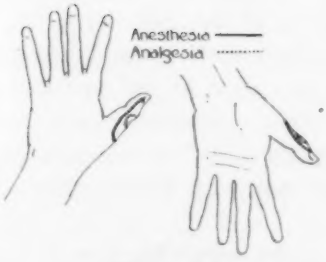


Figure 35

Figure 36

Fig. 35 (Case 27).—Area of sensory disturbance.
Fig. 36 (Case 28).—Area of sensory disturbance.

with subsequent gradual improvement. After one week the only sensory loss was over an area on the thumb about as large as at present. A musculospiral motor paralysis persisted for three months but at present his only marked motor loss is his inability to extend the thumb and index finger.

When examined, June 30, 1919, there were analgesia of the dorsum of the first and second phalanges of the thumb and anesthesia of the dorsum of the second phalanx (Fig. 36). No other sensory loss could be elicited.

SENSORY PHENOMENA

The literature in existence in reference to the sensory phenomena in musculospiral injuries is surprisingly limited, but in a general way there appears to be an agreement that the musculospiral is chiefly a motor nerve and that the sensory changes resulting from its injury are limited in extent and unstable in their location. Head and Sherren[2] even state, "It has long been known that division of the musculospiral in the region of the spiral groove causes no definite loss of sensation over the thumb or back of the hand," and work on the hand and arm was done by Head after division of the external cutaneous nerve as well as of the superficial radial.

Borchardt[3] reports seventeen cases of injury of the musculospiral nerve. In six instances there is no statement as to sensory deficiency. In three cases there was definite sensory change and in the other eight cases the sensory condition is dismissed with such statements as: "Nothing of consequence in the way of sensory loss," "no true sensory disorders," or "a dull sensation in the fingers."

Tinel,[4] in speaking of the subject, says: "Anesthesia in musculospiral paralysis is not often found anywhere else than on the hand. Seldom does it reach the typical distribution of the musculo-spiral, far more seldom does it go beyond it. Most frequently it limits itself to a very restricted part comprising the dorsal region of the first and the second metacarpal. It is often very slight, sometimes even it can scarcely be seen; in any case, it is exceptional to find complete anesthesia in this region; at most we have more or less marked hypesthesia."

Byrne's case[5] showed both epicritic and protopathic loss over the dorsum of the thumb, part of the thenar eminence, the radial half of the dorsum of the hand and the lower wrist.

My own observations lead me to believe that though a lesion of the musculospiral nerve at any point produces less sensory disturbance

2. Head and Sherren: Brain **28**:177, 1905.
3. Borchardt: Bruns Beiträge **97**:253.
4. Tinel: Nerve Wounds. 1918, p. 107.
5. Byrne: J. Nerv. & Ment. Dis. **43**:489, 1916.

than one would expect considering its anatomic distribution, it is rarely, if ever, that some definite area of sensory loss may not be found if carefully sought.

Sensory phenomena, as appreciated by the patient, are not pronounced and are limited in extent. Usually he will tell of an area of numbness over the dorsum of the thumb and especially of the first phalanx, over the first interosseous space and perhaps over the first phalanx of the index finger and more or less over the distal portion of the second metacarpal. The area on the back of the forearm is often ignored even when by examination a large area of well defined sensory change can be demonstrated. In the one instance of well marked sensory change on the back of the upper arm that came under my observation, this was recognized by the patient. A possible satisfactory explanation for the failure of the patient so many times to recognize his sensory loss may be that the musculospiral nerve, limited in sensory fibers, supplies only epicritic and protopathic sensibility to the integument and that deep sensibility is supplied by other nerves. The patient, depending largely on some form of irritation that gives a response through deep sensibility, for his consciðusness of a stimulus, fails to recognize the sensory loss until it is shown him by adequate methods of examination.

In any case, when sensibility is to be tested in musculospiral injury, the skin should be shaved, as was done in all the cases reported, and tactile sensation tested with cotton, wool or a camel's hair brush with the lightest possible touch. Pin prick is usually lost as pain in a similar or somewhat smaller area, but is ordinarily recognized as pressure since, as already stated, deep sensation is not commonly lost in musculospiral injury. Sensation to cold was usually impaired or lost in an area corresponding more or less to the loss for touch and prick and sometimes in a larger area. In some cases the failure to show in the diagram any loss of sense of cold is so out of keeping with the other phenomena that it can be explained only by lack of care on the part of the examiner or by lack of intelligence and attention on the part of the patient. In only one instance was there any excessive response to cold.

Sense of the position of the joints was not lost or notably impaired in any of my cases, but vibration sense was often impaired though not lost, especially on the thumb. Discrimination of two points as two was much diminished in most of the cases, but appreciation of weight was not greatly reduced even in cases with well marked loss of superficial sensibility.

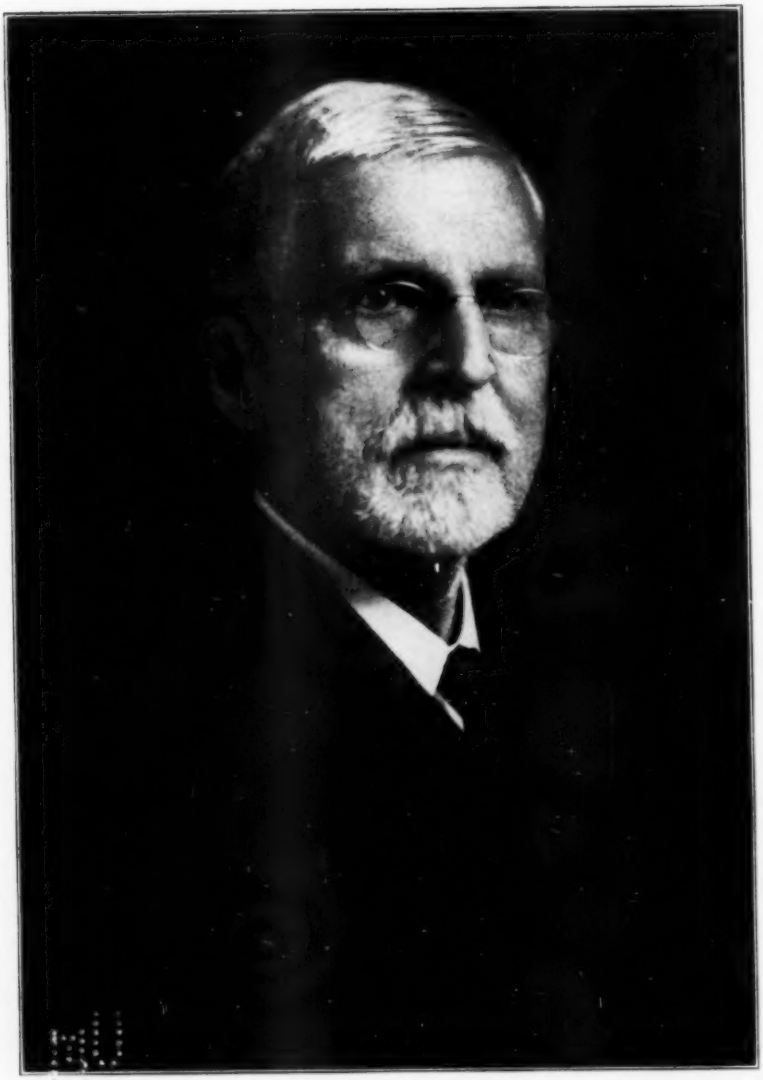
One of the problems which I encountered was to determine the sensory loss in the forearm which can fairly be ascribed to the musculospiral nerve. In several of my cases in which there was no evi-

dence that the musculocutaneous nerve had been severed there was a marked loss of sensation over the area usually ascribed to that nerve. In two instances nearly all of the dorsum of the forearm was numb, but the volar surface was not involved. In such a case it is as reasonable to assume that because the volar surface was little or not at all involved the sensory loss could not be ascribed to any injury to the musculocutaneous or the internal cutaneous nerve as to assume that because the dorsal portion of the area usually ascribed to these nerves was involved, the nerves themselves must be assumed to be involved in the injury. In such cases as Cases 7, 8 and especially 12, it may fairly be doubted that the area of sensibility loss represents only musculospiral involvement; however, it must be understood that no case was included in this series in which the motor phenomena did not clearly demonstrate musculospiral lesion or in which the motor phenomena showed any additional material lesions. A full description of these motor phenomena could only have lengthened this paper.

In those cases in which some degree of numbness was found in the forearm, there was often an area over the dorsum of the wrist in which sensation was more nearly normal or wholly normal. In ten instances in the series reported there was a strip passing from the web between the thumb and first finger in a proximal direction, of normal reaction or of very slight sensory loss separating the more deeply affected area over the dorsum of the first and second metacarpals, respectively.

As a rule, loss of sensation, if present in the thumb, extended down to the nail, but in a few instances there was a narrow band of retained sensibility proximal to the nail, and it was a common occurrence to find the dorsum of the first metacarpal less involved than the phalanges of the thumb. On the first phalanx of the thumb loss of sensibility was ordinarily bounded by the hair line on the median side, but on the external side it extended over somewhat on the palmar surface. This was much more marked over the thenar eminence where the loss of sensibility extended in a curved line on to the palmar surface. Where no evidence of this loss over the thenar eminence appears in a number of the diagrams the explanation probably lies in a lack of care on the writer's part in recording the result of the examination rather than in its actual failure to appear. Where loss of sensibility existed in the area between the thumb and first finger and between the first and second fingers the line of demarcation extended well beyond the hair line and almost to the edge of the web.

I wish to acknowledge my indebtedness to Miss Ethel Cornell, Ph.D., and to Miss Margaret Stubbs for their very valuable assistance in the examinations which form the basis of this paper.



JAMES JACKSON PUTNAM

1846-1918

JAMES JACKSON PUTNAM

HIS CONTRIBUTIONS TO AMERICAN NEUROLOGY

E. W. TAYLOR, M.D.
BOSTON

Dr. Putnam was born in Boston, October 3, 1846, the son of James Gideon and Elizabeth Cabot (Jackson) Putnam, a distinguished heritage on both sides with the very best New England traditions of loyalty and good citizenship. His paternal grandfather, Samuel Putnam, was for many years a judge of the Supreme Court of Massachusetts; his father was a physician of distinction, and his mother a daughter of James Jackson, one of the most notable figures of his period in American medicine, an appreciative biography of whom Dr. Putnam published in 1905.

Dr. Putnam graduated from Harvard College in 1866, at the age of 20; he stood high in his classes, and already gave promise of the attainments of his later years. After leaving the Harvard Medical School, and completing his apprenticeship at the Massachusetts General Hospital, he continued his medical studies in Leipzig and Vienna, under the tutelage of Rokitansky and Meynert. Paris and England were included in his tour of study, and at this time he came into intimate contact with Huylings Jackson, who doubtless exerted a strong influence on his development, and for whom he always entertained the warmest admiration. On his return to Boston he entered on his chosen work with the zeal and enthusiasm of a pioneer, and at once identified himself with the almost untried field of nervous disease. His first appointment at the Harvard Medical School was as lecturer on the application of electricity in nervous diseases—a strange title, illustrative of the conservatism of the time; two years later his title was changed to lecturer on diseases of the nervous system. In 1875, he was appointed clinical instructor; in 1885, instructor, and in 1893, professor of diseases of the nervous system—a position which he retained until his retirement by reason of age in 1912, when he was made professor emeritus. During this long period of activity, he taught and worked unceasingly, chiefly at the Massachusetts General Hospital, establishing there one of the first neurological clinics in the country, ably assisted for many years by Dr. George L. Walton. There were frequent discouragements; the importance of the subject was slow of recognition; the facilities for work were inadequate; prejudices, not yet wholly overcome, were many and disturbing. Dr. Putnam, as one of his contemporaries has recently expressed it, was not a "fighter," but in spite of discouragements the clinic grew; patience and an unfailing belief in the importance of his work

brought results which mere aggressiveness might well have failed to accomplish. By degrees men were attracted to the clinic, and at the time of his death he had the satisfaction of knowing that his early effort had developed into a dignified department of essential and recognized value in the hospital organization.

During the earlier years, after his return from Europe, finding a lukewarm interest in the pathologic anatomy of the nervous system, he established a neuropathologic laboratory in his own house, and there, with an enthusiasm which atoned for lack of proper facilities, he accomplished much of his early pathologic work. This humble laboratory may well be regarded as the forerunner of the present endowed neuropathologic department at the Harvard Medical School. At this time, Dr. Putnam was also much interested in the physiology of the nervous system, in which he found a ready sympathizer in his friend, the late Dr. Henry P. Bowditch, for many years professor of physiology at the Harvard Medical School. His interests were wide, his capacity for work insatiable, and for nearly fifty years his pen was as active as his mind. His papers on a wide diversity of topics within his chosen field were never superficial, and never written merely for the sake of publication. He felt that he had a message, and that it was one of his functions to promulgate knowledge of the nervous system and its diseases in a somewhat unreceptive profession. He wrote assiduously and even laboriously; he was rarely satisfied with the ultimate product, but a reading, especially of his later papers and his addresses, can leave no doubt that he was a rare master of good English. His interest in the classics and in the best literature, together with his general scholarly instincts, combined to give his publications a literary excellence, too often lacking in technical writing. He was peculiarly happy and sympathetic in his biographical sketches, which his natural loyalty to his friends and his keen appreciation of fine qualities of character frequently prompted him to write. Weir Mitchell, Wadsworth, Bowditch and Folsom are some of those whose life-work he gracefully commemorated.

As a teacher he was less successful than as an investigator and clinician. This arose from a certain lack of clarity of expression and an overconscientious attempt to impart to elementary students the profundity of his own knowledge. The result was a failure at times to differentiate essentials from the more unimportant details, which led to a certain natural confusion in the student's mind. To those of more experience in the subject, he was always helpful, stimulating and suggestive.

It is fitting to review in some measure his contributions to medicine and neurology, in spite of the fact that too little time has elapsed to appraise with any degree of justice the full significance of his later

work. A characteristic which stands out prominently was his progressiveness. He never stagnated; interest in the past lay for him in its significance for the future; his zeal for progress and his optimistic hope in new discovery were the keynotes of his energetic life and character, and these qualities are distinctly reflected in his published work. He was almost childlike, and at times possibly unjudicial, in his eagerness to incorporate new ideas into the timeworn problems of disease. This openmindedness, more often seen in youth than in maturity, seemed to increase with the passage of the years and constituted one of the unique charms of his personality.

Fresh from his contact with the leading minds of Europe, his earlier work was concerned largely with matters which were then in the forefront of investigation: localization, the physiology of the cortex, mechanical therapy, animal experimentation, disorders of the peripheral nerves, syphilis in relation to tabes and the like. To all of these subjects he made contributions of value. In 1879, he took up the cudgels for women, who were then under a storm of criticism at Zurich, where they had been admitted to the study of medicine. Much as he disliked controversy, he was a vigorous champion of what he regarded as principles, and then as always he stood on the side of liberality, and took sharp issue with those who saw disaster in the higher education of women and the moral catastrophe which coeducation was assumed to entail. The same spirit was later shown in the vivisection agitation, into which he entered with natural reluctance, but with fixed determination to uphold its justice and necessity.

The first significant scientific contribution was his paper published in 1881, on "Paresthesia of the Hands," probably the earliest adequate description of a condition which has since become generally recognized and incorporated in the textbooks. A few years later he published one of the first descriptions of adult poliomyelitis with the pathologic findings. At this period also he became interested in the problem of lead poisoning with its accompanying neuritis, and prosecuted a long series of researches, which did much toward establishing this important subject on a firm scientific basis. A little later he took up the arsenic question, and pointed out the hidden danger of poisoning from this source. Wall papers were incriminated, whether rightly or wrongly may perhaps be doubted, but this research excited wide interest and something approaching a panic, at least in the vicinity of Boston. In general, it will be acknowledged that this vigorous campaign in industrial disease was a contribution of distinct value and a precursor of the widespread agitation at present in the focus of attention.

Passing over many papers of minor importance, another definite contribution was the paper published in 1891 on "System Diseases of

the Cord," a subject to which Dr. C. L. Dana had almost at the same time devoted attention, and which very properly is associated with the names of both investigators. This again was an original contribution of far-reaching importance with which American neurology should be credited. Myxedema, acromegaly, exophthalmic goiter and the problem of the internal secretions in general were the subjects of various papers, which are of great interest as foreshadowing present day researches. They are all illustrative of Dr. Putnam's tendency to be the first in the field and to play his part in the elucidation of new problems as they arose. In 1895, we find a paper entitled "Remarks on the Psychical Treatment of Neurasthenia," which seems to mark the beginning of his absorbing interest in the psychotherapeutic movement. In this paper, he made an ardent appeal for the recognition of the psychical element in the treatment of what we have since termed the psychoneuroses, but it was many years before he devoted himself almost exclusively to this field of research. Neuralgia of the trigeminal nerve, migraine, hyperostosis cranii, tumors, encephalitis, splanchnoptosis, hydrotherapy, epilepsy, the traumatic neuroses and the surgery of jacksonian attacks are some of the many subjects which from time to time occupied his attention. These papers were all carefully prepared, and helped in greater or less degree in clarifying the unsettled problems of etiology and treatment. In passing, two notable addresses should be mentioned: one, the Shattuck Lecture before the Massachusetts Medical Society in 1899, with the suggestive and happy title, "Not the Disease only, but also the Man," a charmingly written paper embodying the full duty of the physician, as a healer of the mind as well as of the body; the other, a thoughtful contribution to the broader aspects of neurology, read at the International Congress at St. Louis in 1904, entitled, "The Value of the Physiological Principle in the Study of Neurology."

Although the subject of structural neurology occupied a large part of Dr. Putnam's active professional life, he always had the warmest interest in the more subtle problems of the so-called functional disorders. His mind was naturally analytical and philosophical, and he had an overmastering desire to formulate what might be termed a practical metaphysics. "Physics," he said, "can come to its rights only through metaphysics." The attempt to work out a theory of life which should make a rational appeal to the scientific imagination and yet satisfy the fundamental demand for a moral conception of the world was the keynote of his later work. He was doubtless much influenced in his philosophical views by his friends William James and Josiah P. Royce, and by the writings of the German schools of philosophy, from Kant on, and also by Bergson, whom he was always fond of quoting. It is, then, not unnatural that he should have been strongly attracted

by the work of Freud and his associates, which made an immediate and insistent appeal; for the last ten years of his life, as his papers, about twenty-five in number, attest, he gave himself over with his characteristic enthusiasm to the task of reconciling the materialism, or pessimism of Freud, as Southard would have it, with his own unquenchable optimism and with the moral purpose of the world, in which he so thoroughly believed. When the heat of controversial discussion has cooled and his papers are read without prejudice and in the spirit in which they were written, it is my belief that a contribution of no small value to the questions which have always vexed the minds of thinking men will be disclosed. In estimating these last years of his life, it is well for us to realize that he was concerned with no small issues, that he was striving with such help as he could get to work out a satisfactory theory of life in which the conflicting claims of exact knowledge and of philosophy and religion might in some degree be brought into accord—a difficult and perhaps impossible task, but one altogether worthy of the best endeavor of thoughtful men. His last published article, on which he was working at the time of his death, he had entitled, "Strength and Weakness in Psychoanalytic Doctrines." Concerned as he was with what he regarded as the unjustifiability of applying the doctrine of causality in the realm of human thought, as in the realm of physics—a basal principle of the Freudian conception — he says:

> But whatever one may say on this point, it is certain that the chance, made possible by researches of Freud and his colleagues, to substitute knowledge of some sort for unreflective emotional reaction, and thus to eliminate passion, misunderstanding and misery, even if only in somewhat greater measure than before, and withal, to look more deeply into the motives of men no longer living, came to me, as to many persons, as a refreshing breeze.

His misgivings as to the shortcomings of Freud in the sphere of ethics is admirably set forth in his recently published book on "Human Emotion," which summarizes his views on the ethical problem, while acknowledging his full debt to the psychanalytical method of approach.

Believing as he did in the positive contribution of Freudian psychology, he was often distressed by what he regarded as illiberal criticism and misinterpretation; but in spite of occasional outbursts of indignation, unusual in him, he remained tolerant, receptive and generous-minded to the end. His convictions were strong, but he was always willing to hear and profit by the opinions of others, and he combined in unusual degree the capacity for independent thought and sympathetic understanding of others' point of view. He was never dogmatic.

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were, in addition to Dr. Putnam, Drs. William A. Hammond, Roberts Bartholow, Meredith Clymer, J. S. Jewell, E. C. Seguin and T. M. B. Cross. During his forty-three years of membership, Dr. Putnam was a faithful attendant at the meetings of the society, and his name appears many times during these years as a contributor and as a kindly participant in the discussions. His record in this respect perhaps does not equal that of his active contemporary and friend, Dr. Mills, but still stands out conspicuously as a characteristic example of his loyalty and devotion to any organization of which he was a member. He served as president of the association in 1888.

His society membership was large, among them being the American Academy of Arts and Sciences and the Association of American Physicians. Before the latter society a number of his earlier papers were read, but during the later years he was inclined rather to contribute to the meetings of the Psychopathological and Psycho-analytical Association, where he might expect a more sympathetic audience. He was peculiarly sensitive to criticism which savored in the least of flippancy; he was, in fact, a poor controversialist and was devoid of a certain sense of humor, which had he possessed it, might have helped him over many hard places. He had many friends but few intimates; he was, however, essentially a social person and keenly enjoyed contact with his fellows, as many of the older men will attest. In a notice of Dr. Weir Mitchell, whom he greatly admired, he speaks feelingly and modestly of their meeting when he was still a young man "at the foot of the ladder," and of Dr. Mitchell's kindness to him on that occasion, "dining at his table while still in the greenness of relative youth and eating there the fattened terrapin." Speaking of Dr. Mitchell's vigor up to the end of life, he writes of him what might well be applied also to himself:

> So predominantly characteristic was this endurance, until the last, of his vivacity, his eagerness and his activity, that one saw at once in him a man of the type made deathless by Tennyson's "Ulysses," longing, even in his closing years, to re-live the days that had been so full of joys and combats among his peers and seeking to the very last new friendships and new worlds.

With Sir William Osler he stood on terms of intimate friendship, a feeling which was cordially reciprocated. He counted among his closer friends Dr. Henry P. Bowditch, Dr. Oliver F. Wadsworth, and especially Prof. William James, with whose habit of thought he had much in common. But in greater degree than with most men he reserved his more personal feelings of intimacy for his immediate family and for the wide circle of his relatives with whom there existed a peculiarly happy sympathy and companionship. He married rather late in life Miss Marian Cabot, a daughter of Francis Cabot of Brookline and Boston, and Louisa Higginson Cabot. His wife, two

daughters, one of whom is a student of medicine, and a son survive him, the latter a physician.

It is difficult and perhaps unnecessary to speak at length of his wider life in the community in which he and his family always occupied a position of unique respect. His kindness and consideration for the poor and afflicted knew no bounds; it was natural for him to make personal sacrifices for those less fortunate than he, and it was equally natural for him to conceal his well-doing under a cloak of extraordinary modesty and self-abnegation. Active as he was in his professional work, he found time for divers good causes; it was hard for him to refuse any task which he felt it his duty to perform. He was a prominent and active member of the Society of Mental Hygiene, and gave to it much thought and attention. The associated charities for years demanded and received a goodly share of his time. On the inception of the social service movement at the Massachusetts General Hospital, he at once became identified with its activities, was a member of its executive committee, and gave freely of his time and resources to further its interests. Any movement, in fact, which made a human appeal and which offered the slightest opportunity of helping his unfortunate fellowmen enlisted forthwith his warmest sympathies. It may be said that he deserved small credit for this, since his attitude was a distinctly family characteristic, exemplified also in his brother, the late Dr. Charles P. Putnam, and in his sister, now well over 80, who finds her age no barrier to the prosecution of manifold good works. Be that as it may, the fact remains that Dr. Putnam served his community with a disinterestedness which finds few parallels.

Apart from the strictly professional activities which occupied a large share of his attention throughout his long life, he found time for recreations of a simple sort, into which he entered with the same zest that characterized his more serious work. For many years, he maintained a camp in the Adirondacks with his friend Dr. H. P. Bowditch, known familiarly as the Putnam-Bowditch camp, to which he journeyed yearly during the month of September. There he was accustomed to entertain his friends. Were the history of this camp ever to be written, it would reveal the presence from time to time of many notable men who shared and delighted in its cordial hospitality. Dr Putnam also had a summer place at Cotuit on Cape Cod, where his garden and sailing, and his friends divided his attention. Even in his recreations, however, it was hard for him to lay aside the more serious affairs of life. He was apt to have a book in his pocket, and conversation could not long be kept on minor themes. His interest in nature, and in fact in everything animate and inanimate, was extreme; he enjoyed walking particularly, and was quite able to indulge this pleasure with vigor until very shortly before his death; and even then, the warning of his

friends and family deterred him rather than his own sense of incapacity.

His attitude toward the war was characteristic. He was wholly unreconciled to its occurrence; it was constantly in his mind, to the detriment of sleep and of personal pleasure; he seemed to feel toward it a certain personal responsibility — that somehow he was in a measure to blame for the evil deeds of his fellowmen. He had small capacity for lightheartedness when such events were possible in a world which he was forever trying to idealize. He was in a sense a dreamer, and always an incorrigible idealist; tolerant as he was of the opinions of others, he found smallmindedness and narrowness of vision impossible to understand. He was sympathetic with human frailty, but not with bigotry or wilful misdoing. His religion was not so much one of profession as of action; he believed deeply in the beneficent ordering of the world and did his share cheerfully toward trying to make it better and toward reconciling its apparent contradictions and inconsistencies. Men are indeed rare in any community who combine in such superlative degree as did Dr. Putnam devotion to family and friends, a sense of civic responsibility and professional attainment. He was, as President Lowell wrote, in officially commemorating his death, "a man of science eminent in his field, a philosopher and a saint."

His death was sudden. While apparently recovering from a severe anginal attack, he was about to welcome his physician, who had been hastily summoned. The effort was too much for his overburdened heart, and he died with hand extended in friendly greeting, symbolic of the graciousness of his life. He had known for a year or more of his precarious condition, but this in no way abated his activities, which seemed rather to increase than diminish as he knew the end to be approaching. The day before his death he had spent in the open with friends at Concord. His unpublished manuscripts give evidence of his unfaltering mental activity up to the very end. It is a commonplace to say that sudden death is to be desired, nor indeed is this always true; but with Dr. Putnam more than with most men, a period of waning intellectual vigor would have been a calamity, and his family and friends were thankful that he was spared this ordeal.

From his earliest years of practice he devoted himself to his chosen field of work with unflagging zeal and industry. It is hard for us of a later generation to realize the difficulties of establishing neurology in hospital and medical school as a recognized and deserving branch of medicine; he was one of a small and earnest group of men who felt that the time had come for an intensive study of the nervous system in this country, and to him and his contemporaries, some of whom happily are still active in our midst, the younger generation owes a distinct and lasting debt of gratitude.

ELMER ERNEST SOUTHARD

1876-1920

Obituary

ELMER ERNEST SOUTHARD, M.D.

The announcement of the death of Dr. Elmer Ernest Southard comes as a great shock to a host of friends and to American neurologists and psychiatrists. He died in New York City, Sunday, February 8, after a two days' illness with pneumonia. He had gone to New York on Sunday, February 1, to attend the annual meeting of the National Committee of Mental Hygiene and to give an address before the New York Neurological Association. Thursday night he became ill, pneumonia and septicemia developed, and he died the following Sunday.

Elmer Ernest Southard was born in Boston, July 28, 1876. He was the son of Martin and Olive Wentworth (Knowles) Southard. His earlier school training was obtained in the public schools of Boston, and his preparation for college at the Boston Latin School. He entered Harvard College in 1893 and received the degree of A.B., magna cum laude, in 1897. He attained final honors in philosophy, and honorable mention in natural history. In his undergraduate period he was deeply interested in philosophy and psychology. He devoted much time to the courses in logic by Professor Royce, and the foundation he developed there appeared in the mental attitude he maintained in his teaching and research activities in his medical career. It gave to him a habit of clear thinking and an unusual charm of expression that made his contributions and ever ready discussions hold his hearers with keen interest. Even during the busy years of his medical life he found time to attend the seminaries of Professor Royce, and a number of his contributions had their origin in some paper he had prepared for a seminary discussion. This interest in logic always kept him interested in classifications and diagnostic schemes, and determined his method of attack on problems of psychopathology.

In college he was famed as a chess player, and was the holder of an intercollegiate chess championship. His interest in the game was maintained throughout his life, and was for him a source of pleasurable relaxation.

The year of his graduation, 1897, he entered the Harvard Medical School, from which he graduated in 1901. He became intern and assistant in the pathologic department of the Boston City Hospital.

There, under Professor Mallory, he was one of a group of young men of unusual spirit, whose later careers have become conspicuous in teaching and research.

It was at this time that Southard's interest seems to have been directed toward neuropathology. Interest in this subject had been much stimulated by the recent contributions of Carl Weigert to the neuroglia; and the more recent work by Mallory led him to become interested in the problems of the neuroglia, a field in which he always was keenly interested and to which he contributed some of his best work.

In the spring of 1901 he went to Europe and worked for some months with Carl Weigert at the Senkenberger Institute in Frankfurt. From there he went to Heidelberg, where he attended the clinics of Kraepelin and worked in the laboratory of Nissl. On his return to Boston in the fall of 1902, he resumed his work in pathology at the City Hospital. He received the degree of A.M. from Harvard College in 1902. His first medical contribution appeared soon after this, being a study with W. R. Brinkerhoff on "Erythroagglutinins in a Cyst Fluid."

In 1904 Southard was appointed instructor in neuropathology in the Harvard Medical School; he held this position until 1906, when he became assistant professor of neuropathology. Too much cannot be said of the excellence of his course in neuropathology. He brought to it ingenious methods of instruction, and with great success builded the foundations for what later became a separate department of neuropathology in the medical school. His methods of instruction were embodied in his "Outlines of Neuropathology," published in 1906.

Following his return from Europe, Southard continued the interest he had developed in psychiatry while at Heidelberg. He was a most welcome and frequent visitor to the nearby State Hospital for the Insane at Danvers. There he took a keen interest in the work of the laboratory and in clinical visits with the staff to the wards. He thus established between the Danvers hospital and the neuropathologic department of the Harvard Medical School a voluntary but close cooperation in neuropathology that continued for many years.

In 1906, while still assistant professor of pathology, Southard was appointed pathologist and assistant physician to the Danvers State Hospital. Here, with abundance of material, he was most active in research. His alert mind found problems for study in a wide variety of subjects. His unusual capacity for stimulating those who came in contact with him led to a marked productivity in laboratory and clinical studies that continued for some time after he left the hospital. In the three years he was at Danvers his name appears as author or co-author of more than twenty-five contributions. The larger number

of these were neuropathologic. An epidemic of dysentery in the hospital furnished material for several important papers on the bacteriology of that disorder. It was at this time that he was associated with Dr. F. P. Gay, and for a while he worked on problems of anaphylaxis. The results of this were published in a series of papers appearing in 1908 and 1909.

In 1909, through the generosity of Dr. W. N. Bullard, Harvard Medical School established the Bullard professorship of neuropathology, and Dr. Southard was chosen to fill the chair. In this year he resigned his position at Danvers to become pathologist to the Massachusetts State Board of Insanity. His new field of work gave him supervision of the scientific work in all of the state hospitals for the insane in Massachusetts. He inaugurated a close cooperation in scientific investigations of psychiatric problems that has continued to this day.

For several years he, with others, had been greatly interested in the establishment of a psychopathic hospital in Boston. In 1909 a legislative appropriation made this possible, and from then until its opening in 1912, Southard devoted much energy toward planning for the new organization, of which he was to have charge.

In 1911 he was seriously ill from an infection acquired at a necropsy. To recuperate from this he spent a few months abroad, traveling in England and Switzerland. He returned in good health, and in 1912 took up his work as director of the Boston Psychopathic Hospital. He now had fields of work that provided great possibilities for his unusual talents. In all, his energetic personality found some problems that interested him. His interests had a wide range, but he saw in all that he did a direct relation to the functions of a psychopathic hospital as he conceived it. This conception has been embodied in several of his contributions, and was never lost sight of. It was that "the psychopathic hospital should be a center of scientific investigation into the nature, causes and treatment of insanity." He formulated these purposes in programs, and the systematically compiled reports of the scientific work done under the joint cooperation of the State Commission of Mental Diseases and the Boston Psychopathic Hospital show how well his programs were adhered to.

He was always able to attract workers around him, and his personal influence on these will be one of his greatest gifts to medical progress.

There is one special field of work that will be inseparably connected with his memory. This is the development of psychiatric social work and nursing. He had the conviction that the "range of psychiatric advance should be from a medical to a medicosocial range," and that "the outpatient and social service relations of a psychopathic hospital

must be among its most important functions." The splendid psychiatric social service organization at the Boston Psychopathic Hospital will long stand as a model for other institutions. This interest led to his personal initiative in the organization of the School for Psychiatric Social Workers, established at Smith College in 1918.

His broadened clinical interest is seen in the number of contributions of analyses of group clinical material, and his interest in efforts at the correlation of anatomic changes with clinical symptoms. Along this line, his work on dementia praecox and the Waverly researches in the pathology of the feebleminded form what are probably his best known contributions. The Waverly researches were published in monographic form as Memoirs of the American Academy of Arts and Sciences, and in every way are among the most important American contributions to neuropathology.

A bibliography of his contributions published through his regrettably short professional life embraces more than 150 titles. He was the author of two books of importance, one in collaboration with Dr. H. C. Solomon on neurosyphilis, published in 1917, and "Shell Shock and Neuropsychiatry," which has only recently appeared. In both of these his material is presented in case histories, a method of teaching that always appealed to his logical habits of thought.

His interest in prophylaxis of mental disorders and the field of mental hygiene had convinced him of the value of extending methods of psychiatric social work to problems of industries. He had succeeded in securing the cooperation of a number of large industrial organizations in this work, and had begun a work of great possibilities for good.

During the war, Dr. Southard served as major in the Chemical Warfare Division. He entered this at the solicitation of those in charge of this service, who realized his great powers of personal influence and the benefit these would be to the service in matters of morale. Until then he had served as director of the Boston unit of the U. S. Army Neuropsychiatric Training School, and by his personal instruction prepared many reserve officers for neuropsychiatric work in the Army Medical Service.

His breadth of interest is shown in the number and variety of learned associations in which he held membership. These were: the American Academy of Arts and Sciences; the Association of American Physicians; American Medico-Psychological Association, of which he was president in 1919; American Medical Association; American Association for the Advancement of Sciences; American Neurological Association; American Association of Pathologists; Society of Experimental Biology; the Boston Society of Neurology and Psychiatry; National Association for the Study of Epilepsy; American Psycho-

pathological Association; American Genetic Association; Massachusetts Medical Society; New England Psychiatric Society. He was a director of the Eugenics Record Office and a member of the Board of Scientific Directors of the Bedford Hills Laboratory Bureau of Social Hygiene and of the National Committee of Mental Hygiene.

Since the foundation of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, he was a member of its editorial board. He was on the editorial board of the *Journal of Clinical and Laboratory Medicine*, and of the *Psychiatric Bulletin*.

In 1917 he was honored by the degree of Sc.D. from George Washington University.

In 1906 Dr. Southard married Dr. Mabel Fletcher Austin of Boston, lecturer in Wellesley College, who with two sons and a daughter are his survivors.

Southard was a rare personality. He was by type of mind a philosopher. This gave to his psychiatric interests a breadth of view that led him into widely varying fields of medicine and human thought, and largely determined his method of attack on scientific problems. He was a neuropathologist of eminence, whose contributions are notable additions to science. He was a teacher, brilliant and forceful. As an official of the state of Massachusetts, he gave to the public service great energy, and his stimulating leadership and scientific ideals contributed much to elevate the standards in the care of the insane.

His genial humor, his brilliant powers of expression and his broad intellectual interests leave an ineffaceable memory with those who knew him. He was a type of personality whose greatness was peculiarly felt in its stimulating influence on those with whom he came in contact.

His qualities of mind were such as would have made him eminent in whatever field he might have chosen to work. It is for us of the neuropsychiatric field to feel honored that he chose to be of our number.

ALBERT M. BARRETT, Ann Arbor, Mich.

Abstracts from Current Literature

THE FUNCTIONAL SIGNIFICANCE AND PRINCIPAL SYNDROME OF THE CEREBELLUM. FREDERICK TILNEY, Neur. Bull. **2**:289 (Aug.) 1919.

To abstract this article is to do violence to it. The facts presented are packed so closely that it is difficult to condense them without marring the work. The object of this digest, therefore, is merely to sketch the salient features and to suggest that only a reading of the original can do justice to it.

The article, which takes up the whole August number of the Bulletin, is divided into three parts. The first part is devoted to "A General View of the Evolutional Significance of the Cerebellum," the second deals with its "Functions" and the third with "The Principal Cerebellar Syndrome and Its Variations." A case of congenital cerebellar agenesia is described to illustrate the last part.

Part I.—The cerebellum is one of the three suprasegmental portions of the brain. The differentiation from the segmental parts of the neuraxis lies in the fact that for the purpose of expansion the gray matter of the cerebellum is on the surface surrounding the white, and secondly, that the connections with peripheral receptors are less direct, permitting less immediate reflex action, but greater degrees of correlation.

The cerebellum is phyletically of great antiquity; an analogous structure is even said to appear among arthropods. It is constant in all vertebrates and its complexity increases in proportion as the range of movements of the animal advances from the simple to the complex.

In cyclostomes the cerebellum is but little developed; it appears as two dorsolateral evaginations of the metencephalon with direct connection with the somatic sensory tracts. There seem to be present forerunners of the Purkinje cells as well as prototypes of cellular elements later found in the dentate nucleus. In selachians (sharks and rays) the cerebellum is more developed. One can distinguish a granular and molecular layer and large cellular elements with histologic characters of the typical Purkinje cells. The organ has extensive connections for the reception of impulses from the muscles, skin, ear and eye and the gustatory sense. In teleosts and ganoids the cerebellum is similar to that of selachians, except that the splanchnic sensory nuclei are much larger, due to the greater development of the gustatory organs in the bony fish and ganoids. In amphibia the cerebellum is much smaller because of the more sluggish habits of these animals. The granular, molecular and Purkinje layer, however, are well marked. In reptiles the cerebellum is generally small but its size varies according to the activity of the animal. In birds the central arch is enormously developed and forms the vermis, its size being larger in those that fly than in birds that mainly stick to the ground (ostrich, cassowary).

In mammals the cerebellum reaches its fullest development, the large lateral hemispheres being its most conspicuous feature. But there are four essentials to the mammalian organ, namely: (1) a large and complex central arch—the vermis; (2) enormously expanded lateral evaginations—the cerebellar hemi-

spheres; (3) persistence of the flocculus and paraflocculus in rudimentary form, and (4) the presence of a fully developed dentate nucleus and other medullary nuclei.

The size and complexity of the cerebellum in mammals are in direct proportion to the extent of their motor activities, the primates having by far the most highly developed organ. Among the anthropoid apes those who live an arboreal life show great development of the vermis and upper surface of the hemispheres, while in those that tend to become bipedal, whose forelimbs are freed from the necessity of locomotion and tend more toward skilled coordination, the inferior surface of the hemispheres is greatly developed. This accords with the general conception of cerebellar localization.

"The Generalized Pattern of the Cerebellum in Mammals." A comparative anatomic description of the organ is given, based on the works of Elliot Smith and Bolk Smith, the main point of which is to correlate the different parts of the cerebellum with the functional activity of bilaterally and unilaterally synergic groups of muscles in the head, eyes, trunk and limbs.

Throughout the vertebrate series the cerebellum presents a striking uniformity of internal structure, suggesting a definite specialization of function, the ground plan of which is: (1) an outermost zonal layer; (2) a molecular layer; (3) Purkinje cell layer; (4) external medullary stratum; (5) granular layer; (6) internal medullary stratum. The medullary nuclei consist of the nucleus globosus, lateralis (later dentatus), emboliformis and nuclei tecti or fastigii.

In the primitive cerebellum there are connections with several somatic sensory nerves, notably the fifth, with the vagus, the gustatory apparatus, with somesthetic tracts—the optic and auditory (including the vestibular). The main connections are by way of the brachium anterius and posterius, corresponding to the superior and inferior peduncles in higher vertebrates. In mammals the cerebellar connections are made by: (1) the inferior peduncle which contains the dorsal spinocerebellar tract, secondary connections with the columns of Goll and Burdach and olivocerebellar tract; (2) the juxtarestiform body with the Deiterocerebellar tract and secondary connections with the substantia gelatinosa (trigeminal to vermis); (3) the superior peduncle for dentatomesencephalic (red nucleus) fibers and dentatodiencephalic (thalamus) fibers, also possibly for fibers from the red nucleus to the dentate; (4) the middle peduncle for occipito- temporo-, parieto- fronto-pontile fibers to the cerebellum. (5) the ventral spinocerebellar fibers by way of the lateral aspect of the superior peduncle to the vermis.

"Summary of the Evolutionary Significance of the Cerebellum": 1. The cerebellum is a suprasegmental addition to the neuraxis in the interest of coordination. 2. In its primitive form it correlates all the sensory impulses for the purposes of coordination. 3. In the lower vertebrates the main structure is the central arch (vermis) which exerts a bilaterally synergic control over the axial musculature, and the primitive flocculus and paraflocculus permit limited, independent movements of the tail. 4. With the development of the upper and lower extremities there appeared the lateral hemispheres which provided unilaterally synergic control of the individual limbs. 5. As a consequence, the greater the range of independent movements, the greater the cerebellar control. 6. The greatest range of skilled movements being seen in man, he has the most highly developed cerebellum. 7. The vermis still retains control not only of the bilaterally synergic movements of the eyes, face, tongue, jaws, larynx, neck and trunk but also of the extremities which require such control.

Part 2.—The author reviews the clinical and experimental evidence adduced as to the functional significance of the cerebellum. He details the investigation concerning cerebellar localization and the results of experimental stimulation of the cerebellar cortex. Further, the results of Bárány's clinical experimentations, together with clinical evidence, are quoted in support of the view of exact cerebellar localization. Finally, the work of Mills and Weisenburg is critically analyzed as to the significance of the cerebellum in synergic control.

Based on a study (to be reported elsewhere) made with the kymograph and slow moving picture the conclusion is reached that synergia depends on two factors: First, the establishment and maintenance of synergic muscular units, and, second, the establishment and maintenance of coordination between the synergic units in the performance of complex acts. By a synergic unit the author means a group of antagonistic muscles which, however, act together in the execution of a movement. For instance, in flexion of a part there is not contraction alone of the flexors and relaxation of the extensors, but both flexors and extensors contract together—the former more vigorously, acting as the dominant factor in the movement, and the latter less vigorously, acting as the check element. When there is a dissociation in the synergic contractions of the opposed groups there is overaction of one group, because of the absence of the check element, and asynergia or ataxia results.

Simple synergia is the action as a unit of an agonist and its antagonist. Integrative synergia is the coordination of two or more synergic units for the execution of a complex act. Synergic units have kinetic and akinetic phases, that is, periods of contraction which, in the first, represent production of motion and in the second a fixed position. For the maintenance of the proper synergia both the force and the extent must be accurately gaged.

Integrative asynergia is divided into several types, namely: *appendicular,* involving the extremities; axial, involving the axial musculature; trunkal and axio-appendicular.

An interesting conception of the author is contained in the chapter on "Synergia in the Light of Cerebellar Histology." He suggests that the "climbing fibers" of the single Purkinje cells have to do with the regulation of simple synergia units; the "basket fibers" make possible the grouping of many Purkinje cells, and the "granule cells" correlate a vast number of them. For this reason and also because the primary function of the cerebellum is to exert synergic control for the regulation of muscular patterns, localization in it, while definite, is not so discrete as in the cerebrum.

"Summary Concerning the Functions of the Cerebellum":

1. Synergia is the fundamental cerebellar function.
2. The entire musculature of the body is divided into synergic units.
3. Simple synergia depends on the proper correlation of simple synergic units.
4. Integrative synergia depends on the proper correlation of many groups of synergic units.
5. Asynergia or incoordination is the essential cerebellar symptom.
6. Asthenia and atonia are symptoms secondary to asynergia.
7. Synergic control of the cerebellum is ipsilateral for muscles of the same side of the body, but bilateral for axial and trunkal muscles.
8. Synergic control of appendicular muscles is both bilaterally dependent and unilaterally independent, the latter more prominent in the arms.
9. Cerebellar localization is more diffuse than cortical because the former regulates entire motor patterns of an act while the latter controls its purpose.

10. The superior and inferior vermis control the synergic action of the axial and trunkal muscles.

11. The superior vermis controls the movements of the eyes, neck, jaws, face, tongue, larynx and pharynx.

12. The inferior vermis controls the muscles of the trunk and the bilaterally dependent movements of the extremities, e. g., walking.

13. The cerebellar hemispheres control the synergic action of the extremities in bilaterally independent acts, e. g., movements of one arm alone.

14. There may be separate areas for adduction, abduction, etc., but they are to be regarded as parts of more comprehensive regions controlling the movements of extremities.

Part 3.—The case illustrating the principal cerebellar syndrome is that of a man aged 41, who had showed marked disturbances of equilibrium since childhood. There was nothing in his past or family history to account for his condition. Neurologic examination showed: All deep and superficial reflexes were normal except for markedly pendulous knee jerks. Volitional control was normal but there was impairment of skilled acts due to asynergia. The muscle tonus was normal. His gait was reeling, there being no coordination between the movements of his legs and body. There was marked decomposition of movement in walking—asynergia major of Babinski. He showed equal difficulty in standing either with eyes open or closed. Nonequilibratory incoordination was very marked; past-pointing was spontaneous; dysmetria was shown in grasping, reversal of hands and heel to knee tests; adiadokocinesis; the rebound phenomenon of Holmes and tremor of action were found. Nystagmus was irregular, jerky and constant; the head oscillated on turning; speech was scanning, interrupted or explosive. All the other cranials were normal and the rest of the neurologic, systemic and laboratory examinations were negative. Mental status was normal.

In his interpretation and anatomic analysis, the author explains the motor disturbances by the failure of the proper actions of the synergic units of the body. The pendulous knee jerk is caused by the inadequate relation between the dominant and check elements in the synergic unit for flexion and extension of the leg. The disturbances in equilibratory synergic control, both simple and integrative, were shown in the station and gait The disturbances in the nonequilibratory movements, in the cephalogyric movements, in articulation and mastication, as well as the nystagmus and the tremor, were due to dissociation in the synergic units. The fact that the idiodynamic, tonic, volitional and automatic associative control were normal and that sensation was intact absolved the rest of the nervous system and incriminated the cerebellum as the cause of the ataxia.

The clinical picture is recognized as the syndrome of cerebellar agenesia or the cerebellar syndrome of Nonne. Primary progressive cerebellar degeneration of Holmes (a familial disease) and olivo-ponto-cerebellar atrophy of Thomas are mentioned as likewise showing the cerebellar syndrome. The author details all the signs and symptoms which accompany extracerebellar conditions in order to differentiate the pure cerebellar syndrome from them. He finally summarizes the clinical features of the syndrome. He insists that atonia and asthenia are not the result of cerebellar disease but are the consequences of the involvement of adjacent structures, which is rather opposed to the views of Gordon Holmes as expressed in his recent studies on the cerebellum. The author also demands further observation before he would accept catalepsy as a symptom due to a defect of cerebellar function.

It is rather doubtful whether this summary review of the very extensive paper does justice to it, particularly to the original contribution which the author has made with his conception of the synergic unit. The great value of the paper lies in the detailed anatomic, physiologic and clinical descriptions and in their successful correlation. One is greatly impressed by the scholarship shown in the article, and any one who values the fact that the only way to understand clinical neurology is to stress anatomy and physiology, cannot fail to appreciate the paper. Particularly valuable is the comparative anatomic study of the cerebellum, as it helps us to understand the functional significance of the organ from a phylogenetic point of view.

WECHSLER, New York.

ACUTE IDIOPATHIC OR FULMINATING PSYCHOSIS. C. LADAME, Arch. Suisses Neurol. et d. Psychiat. **5**:3, 1919.

Few mental affections have as intricate a history as acute delirium; up to the present time, writers are far from agreement on this subject and the most divergent opinions are held, which are often apparently equally well founded. The writer prefers the designation of acute idiopathic or fulminating psychosis as describing more appropriately the clinical impression of this fatally terminating malady. A host of appellations have appeared in medical literature to designate related phenomena. These may be divided into two types: first, acute symptomatic psychoses, or acute delirium, which is nothing more than a syndrome appearing in a large variety of mental and physical disorders; second, acute idiopathic psychosis, a clinical entity, which progresses in a fulminating manner to a fatal termination. The histopathology, if not specific, is none the less very characteristic.

The etiology of this disease is unknown; the majority of patients reveal some hereditary taint. Of eight cases of acute idiopathic psychosis observed at Bel-Air, all were females; the mortality was 100 per cent. These patients had reached full maturity, the disease occurring between the ages of 25 and 45. The affection is rare. Its course is almost precisely the same in all cases and is somewhat as follows: there is often a slight prodromal period of fatigue and loss of appetite, but the psychosis, itself, begins brusquely and immediately indicates an affliction of greatest gravity. At the end of the first day, the patient reveals all of the clinical characteristics; exitus occurs almost regularly in the first week, rarely in the second, and in a few isolated instances in the third. Cachexia develops with extreme rapidity. The first day is distinguished from the following by the fact that personal consciousness is fully retained; the patient is lucid and presents no difficulty in orientation. However, there is a marked restlessness and anxiety, which continues throughout the course of the disease. Soon the patient becomes confused, the lucid intervals become shorter and shorter and presently marked hallucinations appear. His attention can be held for but a moment. Consciousness is soon replaced permanently by psychic nothingness. Intense psychomotor activity continues, the movements being elementary and incoherent. The patient absolutely refuses liquids and nourishment, spitting them out with an expression of horror. Doutless this sitophobia is based on delusions of poisoning. An extreme fetor is noted about the patient, which possibly furnishes the basis for hallucinations of taste and smell. There is complete insomnia; the urine is scanty and shows slight traces of albumin and deposits of phosphates; there is an abundant acrid perspiration; salivation is copious and the patient expectorates continually early in the disease. Little by little these excretions dry up; the tongue becomes dry,

black, and hard; the lips and gums are retracted and bleeding; the skin looks dry and withered. There is an odor sui generis, which permits diagnosis almost at once. Cardiac action is rapid but feeble; often there is some elevation of temperature with antimortem exacerbation, and bronchopneumonia, pneumonia, and pulmonary edema are noted. Intercurrent diseases play a minor rôle as a cause of death.

Although it is difficult to attempt an explanation of the pathogenicity of an affection of which the necropsy is practically mute, the author feels that certain deductions can safely be drawn owing to the fact that these patients succumbed to an extreme cachexia in which a third to one half of the weight of the body is lost within a few days. It is a fact well known and supported by ample observation on other diseases, that such a marked and sudden loss in weight brings about a quantitive disturbance of the water balance of the organism. There is a considerable and generalized lypoidolysis with an intense disintegration of lecithin as well as of cholesterin. It has been demonstrated that the cell membrane has a very high lecithin coefficient and plays the rôle of an osmotic membrane. Granting the disintegration of lecithin, the semipermeable cell membrane loses its integrity; as a result, a serious osmotic disturbance appears in which the intracellular and extracellular equilibrium of liquids is lost. To this purely mechanical factor is added the action on the tissues of liquids laden with noxious substances. The organism is thus subjected to an intense dehydration. Thus, although we are ignorant of the primary cause of the malady, we know better the succession of morbid processes, once the disease has been initiated.

Necropsy reveals a strong meningeal and encephalic hyperemia; the ventricles are almost deprived of liquids; cerebral edema is present, the cortex presenting the picture of a marked état criblé. Nerve cells and fibers show marked accumulation of isolated and confluent droplets staining with Scharlachrot. The same picture is noted in the cerebellum. There is a marked chromatolysis and karyolysis, cellular sclerosis, and an intense neuronophagia. With toluidin blue, a beautiful metachromasia is noted in the nervous and neurogliar elements. The neuroglia cells are often completely filled with fatty droplets. The blood vessels are engorged with blood to their finest ramifications; a number of them are obstructed with hyalin thrombi. The endothelial cells are distended with lipoid droplets to the point of complete obliteration of the lumen. The perivascular lymphatic spaces are dilated and filled with lymphocytes, mast cells, plasma cells, and Abbauprodukte. The prognosis is bad, exitus occurring early. The writer is of the opinion that many cases diagnosed as acute idiopathic psychosis, which recover, really belong to the type of acute symptomatic delirium. Treatment is purely symptomatic though it would appear that liquid should be administered in large amounts.

WOLTMAN, Rochester, Minn.

THE ENDOCRINE ORIGIN OF MUSCULAR DYSTROPHY. N. W. JANNEY, S. P. GOODHART, V. I. ISAACSON, Arch. Int. Med. **21**:188 (Feb.) 1918.

The authors have investigated the metabolism in nine cases of muscular dystrophy with the view of determining whether that disease "may originate as the result of dysfunction of the endocrine organs." The cases studied were typical dystrophies; practically all showed evidences of trophic changes in the

nails, skin, hair, etc.; some showed peculiar hair distribution; all showed rarefaction of the skull and long bones. Most of the patients were young, only two were adults. Of the group, eight were males.

Careful determinations were made of the creatinin-creatin metabolism, total nitrogen, blood sugar and blood sugar tolerance curves. The calcium and magnesium metabolism was studied in only one case. The diet was creatin-creatinin free. Daily protocols were made of the urinary findings in twenty-four hour specimens.

A summary of the experimental results showed: (1) marked decrease in the preformed creatinin in the urine; (2) abnormal presence of creatin in the urine; (3) low values of creatinin in the blood; (4) normal amount of creatin in the blood; (5) hypoglycemia; (6) delayed glucose utilization (hourly blood sugar curve). Five out of nine cases showed glycosuria after ingestion of sugar in relatively small quantities (exact weights not given). Total urinary nitrogen was low in all cases, but blood urea nitrogen seemed to be normal in five cases. In one case the calcium and magnesium output was low, indicating a probable retention.

As to the clinicopathologic evidence of the endocrine nature of muscular dystrophies, the authors point to the marked bony changes found in patients suffering from those diseases and try to correlate the studies of others made in cases of endocrine disturbance. Of other changes referable to endocrine disorders the authors mention dryness and brittleness of hair, pigmentations, hypertrichosis, peculiar fat distributions and underdeveloped genitalia as having occurred in their series of cases.

The creatin increase in the urine is shown to be similar to that found in starvation, particularly carbohydrate starvation, and the authors maintain that the dystrophy owes its origin to the hypoglycemia which is found. The presence of hypoglycemia in myxedema, cretinism, dyspituitarism and Addison's disease is mentioned to show by analogy the possible endocrine origin of the dystrophies. To show that the hypoglycemia is not due to disuse atrophy of muscles, the authors studied the blood sugar in cases of chronic arthritis with muscular atrophy and found it normal. Experimental removal of ductless glands demonstrated a hypoglycemia.

The authors explain the muscular atrophy on the supposition that it is due to lack of carbohydrates and point to the deposition of fat in the muscles and elsewhere as a sign of hypoglycemia.

Concerning the question as to what endocrine glands are involved in muscular dystrophy the authors review the case reports of numerous other observers, and remark on the want of uniformity of the conclusions reached. Some cases gave symptoms referable to the pituitary, others to the suprarenals, some to the thyroid, etc. The pineal may possibly be a cause in some cases. In the authors' series the presence of acromegaly in one case and possible pineal shadows in two others may point to the two special glands. However, the authors look on the dystrophies more in the light of a syndrome which is the result not alone of disturbance in one gland but in several endocrine organs separately or coincidentally affected.

The study undertaken by the authors is extremely valuable. The evidence, indeed, is not altogether conclusive, but is striking and suggestive, and as the authors well point out, much further evidence may be adduced by scientific therapy. Unfortunately, most of the present day endocrine therapy is of the hit-or-miss variety, possibly because of ill-understood symptomatology and

unbridled speculation. All the more valuable, then, is the exact scientific investigation furnished by the authors. They have made a distinct contribution to our knowledge of the metabolism of dystrophies.

WECHSLER, New York.

MODIFICATIONS OF THE FINGERNAILS IN PERIPHERAL NERVE LESIONS DUE TO GUNSHOT WOUNDS. Lieut. GUISEPPE CARLO RIQUIER, Assistant in the clinic at the Royal University of Sassari, Riv. di patol. **24**:90, 1919.

Changes in the ungual tissues have not been the object of particular study in peripheral nerve wounds during the war. Examination of many cases has convinced the author that such changes do occur and have a marked diagnostic and prognostic value. Increase in nail growth normally occurs to the extent of about 0.1 mm. a day and would equal about 3 mm. a month. Injuries, particularly of the median and the ulnar nerves, will show either an increase or diminution of growth in some or all of the finger nails. He controlled his studies by cutting all the nails at the same level and by painting them with nitrate of silver and measuring daily the nonstained growth which appeared. He found that in the syndromes of irritation of the median nerve more particularly but also of the ulnar, the nails increase in growth rapidly, even in the so-called cases of causalgia although in this condition all the nails were affected. In lesions showing complete interruption of the nerve the growth of nails was greatly diminished. There were also changes in the color of the nails, opacity, size of the lunula, the striations and changes in the lateral and anteroposterior curves. The author agrees that these lesions are also found after infections of the extremities, injury to vessels and even in lesions of the central nervous system. Changes in color, striations and in the curvature of the nails were found in one case of left radial nerve injury. The author feels, however, that these small nail signs in these cases are due to disuse which influences particularly the curvature of the nail because of the atrophy which occurs in the tips of the fingers which build up and give shape and curve to the nails. As a sign of specific irritative lesion of the median and the ulnar nerves the mere change in the curve of the nail is not important unless it assumes a grotesque form in which case a real ungual hypertrophy is characteristic of median nerve injuries. Here, however, one notes also great rapidity of growth with many longitudinal and transverse sulci. In destructive lesions one never sees marked changes in the curvature of the nails. Indentations and depressions with opacity, variations of color, etc., are invariably pathologic signs of irritative or partial lesions. Another important pathologic sign is the presence of a sharp line between the proximal and distal lamina of the nail which sharply delineates the normal proximal lamina from the changed distal lamina which will show various degrees of change in color, shape and size. This may be indicative of a lesion in both the median and ulnar nerves. The author has been able to show in some cases that changes of this type occurring in the last three fingers were more apt to occur with ulnar lesions. In lesions of the sciatic and internal popliteal, disturbances of the growth and morphology of the nails were also encountered, but their study is made difficult by the fact that variations in the type of normal nails of the toes is great, due to interference and modification caused by footgear.

OSNATO, New York.

THE EXPERIMENT IN OCCUPATIONAL THERAPY AT BASE HOSPITAL 117, A. E. F. SIDNEY I. SCHWAB, Ment. Hygiene **3**:580 (Oct.) 1919.

Schwab presents an interesting account of the experiment in occupational therapy at Base Hospital 117, the A. E. F. war neuroses treatment center. The problem was to return to the firing line, in a relatively short time, as large a proportion as possible of organically sound but functionally defective soldiers. That occupational therapy played a most important part in the healing of these "bloodless wounds" is clearly indicated by the fact that in 85 per cent. of the 3,000 patients who passed through the hospital, occupations of various kinds were utilized as therapeutic agents. The list included stone-breaking, road-building, wood-chopping, tilling, carpentry, tinning, printing, weaving, painting, sketching, wood-carving, etc. The philosophy of work applied to the neuroses, as Schwab sees it, lies in the fact that both the physiologic and psychologic needs of the patients are met and adequately supplied. For instance, even such a simple matter as stone-breaking, exerts a beneficial mechanical effect on the excessive muscular activity which is a concomitant of ataxia and tremors and by the coordinated movements involved favors a tonal balance between antagonistic muscle groups. From the more strictly mental point of view, the patient is impressed by the demonstration of the fact that his illness must be evanescent, since the muscles affected by the physical defect (tremor, palsy, etc.) are capable of performing coordinated movements and are responsive to motor control. Furthermore, work and particularly the more artistic forms of endeavor, supplied an outlet for the emotional hypertension which exists. The author rightly stresses the necessity of regarding occupation as a medicine, which must be individually prescribed by the physician. Neglect of this factor of individuality means paucity of results and possible harm to the patient. The experiment at La Fauche not only proved the feasibility of work as a valuable form of treatment of the war neuroses in the zone of advance, but was further a distinct forward step in the evolution of the occupational therapy idea, which will bear fruit in civil life.

STRECKER, Philadelphia.

ANAPHYLACTIC PHENOMENA IN THE PATHOGENESIS OF CERTAIN EPILEPSIES. P. PAGNIEZ and P. LIEUTAUD, Presse méd. **27**:693 (Nov. 19) 1919.

Starting with an anaphylactic conception of epilepsy, the writers cite one case which can reasonably be viewed from that standpoint. Certain therapeutic tests were applied, and results obtained which were instructive and suggestive within the limits of the investigation.

Widal (*Soc. méd. d. hôp. de Paris*, Feb. 13, 1914) had established an anaphylactic basis for urticaria: certain foods could give rise to cutaneous, digestive, and respiratory disturbances, preceded by blood-vascular changes in the nature of a crisis ("crise hémoclasique initiale"). Other writers had shown that minimal doses of the noxious substance, ingested some time before the meal which would contain it in quantity, prevented both the crisis and the subsequent symptoms. The recognized importance of alimentary disturbances and dietary regulations in epilepsy is cited, and an attempt is made to demonstrate related anaphylactic reactions.

A classical case of epilepsy, which had shown improvement under some dietetic care, and which was not being treated by bromid or other drugs, was

selected for experimentation. Ordinary meals of bread, meat, eggs and vegetables, with a little wine, were not followed by attacks, and examination from hour to hour showed a normal digestive leukocytosis and slight elevation of blood pressure. When 50 gm. of chocolate were added to the meal, it was followed by a series of sharp oscillations in the blood count, dropping to a leukopenia of 3,600, accompanied by some lowering of the blood pressure. This was considered a true crisis ("crise hémoclasique" of Widal), and was followed shortly by one or more convulsive attacks. Several days later, when 0.050 gm. chocolate was administered forty-five minutes before the same type of meal, including 50 gm. chocolate, the blood disturbances and convulsive seizures were absent. Both experiments were repeated several times on the same case, with appropriate controls.

HUDDLESON, New York.

SEVERE POST-DIPHTHERITIC PARALYSIS FROM WOUND DIPHTHERIA. E. RÖPER, M.D., Neurol. Centralbl. **38**:450 (July 16) 1919.

The war offered unusual opportunities to study diseases and injuries of the peripheral nervous system, especially severe multiple neuritis with paralysis of all extremities from furunculosis, chilling of the body, grip, etc., with complete recovery. Neuritis following wound diphtheria occurred more frequently than was supposed, because the diphtheritic infection of wounds was often overlooked, unless the neuritis following exhibited characteristics of diphtheritic paralysis.

An officer, aged 25, was wounded in the left hand by a hand grenade April 27, 1918. The wound was small and deep. He had a fever of 39.8 C. on the fourteenth day, smear-like exudate in the wound, swelling of the hand, discharge of fetid pus, and necrotic tissue. During the seventh week the patient developed bilateral paralysis of accommodation. In the eleventh week there were numbness of the tongue, lips, fingers and legs, weakness of the arms and legs, absence of all deep reflexes and tenderness of the sciatic nerves. There was general improvement with normal neurologic findings by May, 1919. No bacteriologic study of the wound was made.

A soldier, aged 27, was wounded by a bullet in the left thigh March 10, 1918. He became very anemic from loss of blood. No injury to the nerve trunks was found. The wound remained clean until May 21, when fever and wound inflammation with foul exudate developed. On November 2, neurologic examination showed muscular weakness, anesthesia in the legs, ataxic gait, weakness of the reflexes in the arms, absence of the knee and Achilles reflexes, and the presence of the pharynx reflex. The eyes were normal. The diphtheritic character of the paralysis suggested a cultural investigation of the wound, which revealed diphtheria bacilli. In both instances, the diphtheritic character of the wound infection was first suggested by the results of a neurologic examination.

SHELDEN, Rochester, Minn.

THE PRESENT POSITION IN CLINICAL PSYCHOLOGY. WILLIAM MCDOUGALL, J. Ment. Sc. **65**:141 (July) 1919.

An historical account of the theories of the workings of the mind is attempted by Major McDougall. He discusses the sensationism in its combination with associationism which gave us our picture of brain-cells connected to switchboards, and led research workers to neuropathology.

The casting aside of mechanistic psychology is best illustrated by the work of Freud, who has brought two great facts to light: (1) "the impulsive, demoniac, illogical nature of much of human thought and conduct;" (2) the poor representation in consciousness of this impulsion. The over-importance attached to sex by Freud has been partly counteracted by later studies of self-assertion, gregariousness and fear. The author emphasizes that these fundamental impulses, and others, are purposive and that our conduct is to be regarded as determined not only by the events of the past but by ideals of the future. Man "is borne on to his biological ends, for the most part but dimly conscious of those ends and of the mental forces and processes by which he achieves them." The conception of these forces, not distinct from consciousness, but greater than consciousness, is of more value than the vague "unconscious" of Freud and Jung. That the individual mind reacts according to its inherited impulses is a final assumption worth much to medicine because it explains "the uniformity of thinking—revealed by primitive myth and custom" and by the symptoms of certain mental disorders.

The reviewer questions whether mechanistic psychology needs to be hurled so far out of the way of the onrushing "clinical psychology"—certainly the structure of our nervous system is worth some study.

BOND, Philadelphia.

FOREIGN BODIES IN THE CEREBRAL VENTRICLES. G. L. REGARD, Presse méd. **27**:645 (Nov. 1) 1919.

The writer in discussing foreign bodies in the cerebral ventricles limits himself to such foreign bodies as bullets or fragments of shells, and does not include foreign bodies of a different category, such as hydatid cyst. His experience has shown that foreign bodies of the type he considers are most frequently found in the lateral ventricles and least frequently in the fourth. He expresses the opinion that they reach the ventricle secondarily, having first lodged in the adjacent brain tissue. In connection with naming over the symptoms which are the usual ones of brain injury, he warns against lumbar puncture in these cases on the ground that it occasions a diffusion of microbes from the site of the foreign body.

The author relies on the roentgen ray for diagnosis. Radiographic examinations indicate the intraventricular site of a foreign body, though that fact is definitely established only if successive roentgen-ray examinations show changes of location of the foreign body dependent on changes in position of the head. A foreign body can be, as it were, "tracked" into the frontal or temporal extensions of the lateral ventricle. A foreign body not displacable by the head movement but "caught" in the middle ventricle, is diagnosed on presumption when its roentgen-ray shadow lies exactly beneath the sagittal suture, is 25 mm. above the zygomatic arch and from 0 to 20 mm. in front of the opening of the auditory canal. Similarly a foreign body, not displacable, owing to its size, is given a presumptive diagnosis of location within the lateral ventricle when it lies within the four classic topographical lines outlining this ventricle.

The writer outlines a two-stage operation for the removal of foreign bodies from the ventricles—the latter part of which is done coincident with radiographic inspection. He gives a good prognosis for such cases if only the complication of infection can be avoided.

DAVIS, New York

Society Transactions

AMERICAN NEUROLOGICAL ASSOCIATION

Forty-Fifth Annual Meeting, held at Atlantic City, N. J., June 16-18, 1919

(Continued from page 208)

21. CEREBELLAR DYSSYNERGIA WITH SPECIAL METHODS FOR ITS STUDY AND ANALYSIS. ADRIAN V. S. LAMBERT, M.D., and FREDERICK TILNEY, M.D., New York.

Illustrated by novographic motion pictures, and kymographic tracings.

DISCUSSION

DR. L. PIERCE CLARK, New York: This extraordinary and interesting method of case study is certainly delightful both clinically and experimentally. I should like to ask Dr. Tilney whether it has been extended to any other type of motor disorder, for instance, myoclonia and epilepsy. I would like to voice my regret that when I studied the convulsive phenomena of epilepsy in 1898 I searched everywhere to find a machine to produce the same thing that Dr. Tilney has shown in such an excellent manner. What was particularly needed there was to take the rapid discharge and muscle movement phenomena of epilepsy and bring them down to slow movements so that one could study the initial foci in the jacksonian type. Since then the moving picture has been developed. This means of getting at the segmental movements and bringing them in for careful study is of enormous importance as it concerns the whole convulsive phenomenon of epilepsy. The teaching value also is considerable as it will give medical students a more definite knowledge of the motor phenomena in all convulsions. I would like to ask Dr. Tilney if in any of his cerebelloid cases instruction would modify this asynergic behavior. The teaching of graceful movements which can be used all over the field of disease phenomena in the selection of better methods of motor coordination, could as well be applied to normal children in schools.

DR. FREDERICK TILNEY, New York: I will say just a word in answering Dr. Clark, with reference to applying this method of study to myatonia and locomotor ataxia. It is an expensive undertaking and the pictures of the cerebellar cases cost several thousand dollars. We spent a large sum of money in getting a limited amount of film. Photographing the epilepsies would be difficult and expensive. As to the education of patients with asynergic movements, that has so far not been practicable, but perhaps later on something of that sort will be reported.

22. CONGENITAL FACIAL PARALYSIS. FRANK R. FRY, M.D., and MICHAEL KASAK, M.D., St. Louis.

This article appeared in the December number of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, page 638. No discussion.

23. HEREDITARY OCCURRENCE OF HYPOTHYROIDISM WITH DYSTROPHIES OF THE NAILS AND HAIR. DR. ALBERT M. BARRETT, Ann Arbor, Mich.

This article appeared in the December number of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, page 628.

DISCUSSION

DR. BEVERLEY R. TUCKER, Richmond, Va.: Dr. Barrett has opened an interesting field in the history of the endocrine disturbances. I recall the case of a family in which the father and mother, both absolutely normal, had six children who were all normal except one and she was distinctly a cretin. When she grew up she married an alcoholic and they had seven children, and of these seven children every child suffered from convulsions. One had one convulsion, two had such frequent convulsions that they were sent to an epileptic colony in Virginia. I was more or less fortunate in being able to get roentgenograms of the pituitary of five; three showed no change in the sella, two showed distinct hypopituitary changes and they were put on pituitary extract. One improved to a fair degree, and the other has not had a convulsive attack for over two years. None of them showed any sign of hypothyroidism clinically though the mother was distinctly hypothyroid, except possibly the youngest child who was 2½ years old the last time I saw it. As to the nails and hair, I am afraid to venture discussion on that because I have not my notes with me. The point I would like to emphasize in these endocrine disturbances is that in the succeeding generation we are likely to get not only the ductless gland disturbance of the parent but other ductless gland disturbances and various forms of feeblemindedness, and we cannot predict the absolute relation. It is interesting to note that the father in the case just recited was an alcoholic.

THE PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 21, 1919

J. HENDRIE LLOYD, M.D., *President*

AN UNUSUAL SYMPTOM GROUP FOLLOWING INFLUENZA.

Presented by DR. BENJAMIN WEISS.

An Italian, aged 30, a stone mason, married, whose wife had had three children and no miscarriages, denied venereal infection, and the family history was negative. He was well until October, 1918, when he had a severe attack of influenza, and was confined to bed for a period of two weeks.

In January, 1919, the man began to have difficulty in walking. He stumbled and had poor control of the movements of his legs. His gait gradually became worse and he required a cane in getting about. He also noted that his vision became less acute, more especially that of the right eye. He had had severe occipital headache for several days without vomiting.

Physical Examination.—The gait was staggering, with a tendency to fall laterally, usually to the left. The incoordination became much more marked when he walked with his eyes closed. The station with the eyes open was poor; there was a tendency to fall either to the left or right, or backward. With his eyes closed his equilibrium was greatly disturbed. The eyes were prominent but had been so since childhood. The pupils were equal and round. The right pupil reacted sluggishly to light, but reacted well to accommodation. On extreme lateral rotation nystagmoid movements were noted. No ocular palsies were apparent. The eye ground examination revealed secondary optic atrophy in the right eye and a choked disk in the left.

The upper extremities showed no loss of muscular power. There was some incoordination on the right in the finger to nose test. His penmanship was poor. Adiadokocinesis was present on the right and absent on the left. The reflexes were normal. The lower extremities showed good muscular power in both legs. In the heel to knee test there was some ataxia in the right leg, but none in the left. Ankle clonus and Babinski's sign were absent on both sides. The patellar reflexes were slightly exaggerated. The sphincters were uninvolved and there were no sensory disturbances. The memory was unaffected. The skiagraph of the head showed no evidence of any lesion; examination of the ears revealed no pathologic changes. The blood and spinal fluid examinations were negative.

FRIEDREICH'S ATAXIA IN TWO COLORED BOYS—BROTHERS.
Presented by Dr. J. Hendrie Lloyd.

Dr. Lloyd said his cases were of interest, not only because they were quite typical, but also because they occurred in colored boys. Friedreich's ataxia in the negro was probably rare; at least, Dr. Lloyd did not recall having seen an instance of it before. The patients were full blooded negroes; and for their ages they were fairly well developed and intelligent. The father, aged 39, and the mother aged 35, were both apparently healthy. The mother and one daughter gave negative reactions to the Wassermann test of the blood. The blood could not be secured from the father; but for the question of possible congenital syphilis the test of the mother and daughter's blood was more important. Both patients gave negative reactions from the blood and spinal fluid. Although there was not much ground for supposing that congenital syphilis was a cause of Friedreich's ataxia, it was thought desirable to test this question as fully as possible. The mother had had five children; the eldest, a daughter aged 14, is living and well; two sons, the patient and another son who had convulsions and died at the age of 3; and a daughter, aged 3, who is living and well. There was one miscarriage at seven months. Otherwise the family history was unimportant. There had been no ataxia in collateral branches so far as could be ascertained.

I. C., the elder boy, aged 11, was born by normal labor. He learned to talk and walk at the usual age. He went to school and did well. He had convulsions at the age of 1, and some of the usual diseases of childhood. The present trouble began, when he was 8 years old, with awkwardness in the leg movements, the patient falling to his knees when attempting to walk. He had a marked scoliosis. The gait was ataxic, the feet were placed far apart and the boy swayed violently in walking, which he did with difficulty, requiring assistance. It was not the gait of locomotor ataxia. The station was very unsteady, and was increased on closing the eyes. The finger to nose test and the heel to knee test showed ataxia; there was marked adiadokocinesis. The knee jerks and Achilles' jerks were absent, and there was no Babinski reflex on either side. The abdominal and cremasteric reflexes were present. Sensation to touch, pain, heat and cold was normal, but there was some loss of sense to a tuning fork placed on the tibia, radius and ulna. No shortening of the plantar arch, or hammer toe, which is described as seen in some cases of this disease, was present, but there was marked flat foot, with slight eversion. No loss of sphincter control; no muscular atrophy; no subjective pain; no girdle sense were found. The speech was slightly hesitating or staccato, not drawling. The eye condition is noted below.

M. C., the younger boy, aged 9, born of normal labor, talked and walked at the usual age, went to school and was considered very bright. He had convulsions at the age of 2 and the usual diseases of childhood. The present trouble was first noticed when he was 6 years old, and was recovering from an attack of typhoid fever. Dr. Lloyd said that the boy's gait and most of his other symptoms were so similar to those of his brother that it was hardly necessary to recapitulate them in detail. There was the same gait, with swaying movements, and unsteady station, which however, was not increased on closing the eyes. The deep reflexes were abolished; there was no pain and no loss of sensation in any of its modes, except for the tuning fork, was detected. There were, however, no deviation of the spine, and no muscular atrophy or loss of sphincter control. The speech was slightly hesitating.

Dr. Shannon reported concerning the condition of the eyes: In the elder boy the vision in both eyes was normal. The pupils were equal. The reaction to light, direct and indirect, was sluggish. Accommodation and convergence reaction was prompt. The iris showed a curious lack of pigmentation in the upper nasal quadrant of the right eye, and in the upper nasal and lower temporal quadrants of the left eye, suggesting iritic atrophy at these points. On distant fixation the left eye showed a tendency to rotate outward, although on close fixation fair convergence was obtained, indicating a condition of exophoria. This measured with the Maddox rod about 12 degrees. The characteristic ataxic nystagmus was not elicited. In the right eye the media were clear; the disk discolored and somewhat atrophic; the margins clearly outlined; vessels moderately contracted; macula normal; no changes in the periphery of the fundus. In the left eye the conditions were similar.

In the younger boy, the vision was about normal. The pupils were equal, and reacted sluggishly to light, but promptly to accommodation. the irises were normal. The characteristic nystagmus, sometimes called pseudonystagmus or ataxic nystagmus, was observed when the eyes were fixed on a moving object, especially in the extremes of rotation. In the right eye the disk was discolored and atrophic and the vessels contracted. In the region of the macula a leaden-hued area, about the size of the disk, was covered by a sprinkling of small round or triangular coal black pigment patches. In the extreme nasal field additional pigment patches were seen, and isolated patches of pigment were seen in various parts of the fundus. The left eye revealed the same condition.

It was noteworthy that neither speech defects nor nystagmus were very well established in these cases, but these defects are sometimes rather late in appearing even in typical cases of Friedreich's ataxia. The appearance of slight optic atrophy was also noteworthy. Several cases have been reported in which the light reflex was lost. Gowers thought this an evidence of syphilis. It may be an evidence that the case was one of juvenile tabes.

FRIEDREICH'S ATAXIA IN MIDDLE AGED MEN—TWINS. Presented by Dr. George Wilson.

Dr. Wilson said that the cases which he desired to present were interesting because they were atypical and occurred in twins.

Case 1.—The elder of the pair was admitted to the Philadelphia General Hospital in the service of Dr. Spiller, Sept. 25, 1919. The man said that he was in good health until he was 33 years of age, twelve years ago. At that time he noticed that he had difficulty in walking; this infirmity gradually increased, but was never so severe that he could not walk. A few months

after the initial symptom the patient noticed that his speech was becoming "thick." As far as the man was concerned, he had no other symptoms. In addition to his twin, reported below, he had two brothers and one sister who were living and normal. The patient had drunk moderately. He denied syphilis but had had gonorrhea.

Physical Examination.—The man was a well nourished Jew. He was cranky, and did not adapt himself readily to his surroundings. Romberg's sign was present, and the gait was an ataxic, rolling one. Marked clumsiness and ataxia in the hands were brought out when the man dressed and undressed himself. Adiadokocinesis was present on both sides, and a slight intention tremor developed in the finger to nose tests. The irises were brown, the pupils were equal, centric and reacted well to light and in convergence. Horizontal nystagmus was present. There was no exaphthalmos or hemianopsia. Dr. Shannon reported that the optic nerves were normal. The cranial nerves were normal. The speech was thick and slurred.

The patellar and achilles' reflexes were present and lively. The biceps and triceps reflexes were present, but depressed. The cremasteric and abdominal reflexes were present; plantar stimulation produced flexion of the toes on both sides. Pain sense, heat and cold were normal throughout. The sense of position was affected in the toes of both feet. There was no paralysis, no muscular atrophy or hypertrophy, no deformities of the spine or feet and no cerebellar catalepsy. The blood Wassermann test was negative; the patient refused to permit a lumbar puncture.

CASE 2.—The second patient was a replica of his brother. The story of his illness and examination was practically the same as the record given above. The second patient was as affable as his brother was disagreeable. He had had both gonorrhea and a chancre although the blood and cerebrospinal fluid were normal. The patellar reflexes in the younger of the twins were distinctly exaggerated without the presence of ankle clonus or Babinski's sign.

Dr. Wilson said that the cases which he had presented were of an unusual type. The symptoms were late in appearing and an analysis of the histories showed many atypical findings. Though the patellar reflexes were exaggerated, this sometimes occurred in Friedreich's ataxia together with Babinski's sign because the pyramidal tract might be degenerated. The deformities of the feet, toes and spine did not always occur and were absent in these cases. Dr. Wilson observed that the cases of Friedreich's ataxia which were presented to the society demonstrated that Jendrassik was correct when he said that the family diseases of the nervous system blended almost inseparably one into the other and that the symptoms were constantly overlapping. Jendrassik also said that a family disease when present in a family might be, and often was, atypical but that all of the members affected were involved in the same manner.

TWO SISTERS WITH FRIEDREICH'S ATAXIA. Presented by DR. J. W. MCCONNELL.

A. H., aged 22, whose walking had been bad since she was 14, was for a time improved by using a prescribed shoe; but of late she had been stumbling with unusual frequency, even falling without apparent cause

Examination showed abolition of the reflexes in the upper extremities with a very insignificant atrophy of the left hyperthenar eminences with distinct loss of power in the lower limbs and almost complete inability to elevate the toes and foot. The reflexes in the lower extremities were completely lost, not rein-

forcible; both feet were instances of moderate talipes cavus, and she had the distinct deformity of the toe which is seen in Friedreich's disease. She showed no cranial nerve conditions, no speech defect, no disturbance of vesical or rectal sphincters, no spinal curvature. She had no ankle clonus or Babinski sign. There was no tenderness over the nerve trunks nor were the nerves palpable. She had no sensory disturbance. The sister of this girl, three years younger, presented a similar foot deformity with lost reflexes in the lower limbs.

Her mother had had difficulty in walking, with a deformity of the foot and was for many years previous to her death quite invalided. The maternal grandfather also had had a foot deformity, but so far as could be learned he had had no paralysis or disturbance of locomotion.

Both these cases were regarded as probable instances of Friedreich's ataxia.

DISCUSSION ON FRIEDREICH'S ATAXIA

DR. W. G. SPILLER stated that the three families presented three different types of Friedreich's ataxia. He thought Dr. Lloyd's cases were typical. The twin brothers shown by Dr. Wilson belonged more to Marie's hereditary cerebellar ataxia type, especially in the age at which the symptoms developed, and in the preservation of the patellar reflexes in one and the exaggeration of these reflexes in the other. The girls presented by Dr. McConnell showed the earliest form of Friedreich's ataxia. They had the distinct Friedreich toe, loss of tendon reflexes, ataxia and nystagmus.

DR. J. H. LLOYD brought forward the question of syphilis as a cause of Friedreich's ataxia. Dr. Spiller said it would be strange that syphilis should affect two members of a family in so similar a degree as in the cases presented. Two cases in each of the three families were much alike, and yet all three types were different. Syphilis in attacking a family so as to produce disease of the nervous system is likely to produce different manifestations —for instance, in one tabes, in another paresis. When husband and wife have contracted syphilis, probably from the same source, the forms of the syphilitic disease need not always be the same.

The optic atrophy and absence of light reflex in Dr. Lloyd's cases occur in the form of the disease described by Marie, which probably should be considered as a part of Friedreich's ataxia.

Friedreich's ataxia is usually regarded as a rare disease, and it was an extraordinary coincidence that three different families showing three different types of the disease should be presented at one meeting. As for being rare in the negro, it follows that it is likely to be so when it is rare in the white race.

From the pathologic standpoint there is much to be said of the relation to one another of several forms of family disease of the nervous system. Family spastic paralysis has pathologic findings resembling those of Friedreich's ataxia. It has been shown that the family form of optic atrophy may be associated with symptoms of posterolateral sclerosis.

DR. CHARLES K. MILLS said he had nothing in particular to add, except perhaps to call attention to the distinct cerebellar features in the symptomatology, especially in Dr. Lloyd's cases. It was well known, however, that in Friedreich's ataxia we had a cerebellar symptomatology, in part at least. Dr. Mills thought that Dr. Spiller had not said very much about this, except in referring to Marie's paper.

Dr. S. D. Ingham said that he had recently seen a case that belonged to the same group as those just presented, although it differed from them in certain points, and belonged rather to the type of Marie than that of Friedreich. The patient was a married woman about 30 years old, who, during the past two years, had gradually developed difficulty in walking. The gait was distinctly of the cerebellar type, similar to that of the negro boys shown by Dr. Lloyd. The tendon reflexes were very active, especially in the lower extremities. Ankle clonus and Babiński's sign were demonstrable on both sides. The attitude of the feet in relaxation was equinocavus with dorsal extension of the great toes to a position at right angle to the tarsal bones. Dr. Ingham thought that this case, considered with those presented, well illustrated the variations in the involvement of the various spinal tracts in this disease. In his case there was positive evidence of pyramidal tract degeneration, in addition to that affecting the cerebellar system.

Dr. J. Hendrie Lloyd said, in reference to syphilis, he did not mean to imply that his cases were syphilitic, but he thought that this was a question that should be thoroughly sifted. In reference to syphilis in some members of a family and not in others, he would remind the members of the old law of the attenuation of the virus. That is to say, the first of a syphilitic family would be very syphilitic, perhaps would be stillborn; the next would perhaps break out in syphilis in six weeks. Later children would show syphilitic infection in lesser degree. Jonathan Hutchinson was an advocate of this law of the attenuation of the virus. Dr. Lloyd said he did not believe much in the law, but it only proved that there can be some children more affected than others in the same family. Cases of twins have been reported, born of a syphilitic mother, in which only one of the two children was infected. The tests have not proved that any of the family were syphilitic in the case of the negro boys. In reference to attitude, one boy had the Romberg symptom pretty well developed, the other not. The appearance of slight optic atrophy in both cases is noteworthy.

HEMIPARESIS WITH PARALYSIS OF THE SYMPATHETIC. Presented by Dr. William G. Spiller.

The patient was a boy in whom a hemiparesis had existed from early childhood, and on the side of the hemiparesis the sympathetic supply of the eye was paralyzed. He discussed the representation of the sympathetic brain. The paper will be published in full later.

DISCUSSION

Dr. S. D. Ingham recalled having seen a patient in whom unilateral sympathetic symptoms were apparently due to a lesion above the medulla. A thalamic lesion was indicated in this case by a disturbance of tactile, pain and thermic sensation together with spontaneous pain, all of unilateral distribution. The associated sympathetic symptoms consisted of a small pupil and a narrow palpebral fissure on the affected side, and unilateral sweating of the face and trunk. There was also a slight edema or puffiness of the hand of the affected side. In this case it was apparent that the sympathetic symptoms were not limited to the distribuion of the cervical ganglions since they extended to the arm and trunk. In Dr. Ingham's opinion this case furnished evidence of what Dr. Spiller had emphasized, namely that there is a definite area for the control of sympathetic functions in the region of the midbrain. He thought that it could be placed at least as high as the basal

ganglions, probably in the thalamus. The question of the cortical control of the sympathetics is a very difficult one. It is well known that the emotions play an important part in causing sympathetic reactions, but these effects are apparently more dependent on emotional reactions than on the stimulation or the loss of any particular part of the cortex. Experimental work along these lines has not been very satisfactory, as it was difficult to get a fair test by which to associate sympathetic functions with cortical representations.

Dr. S. Leopold said he wanted to emphasize what Dr. Spiller had brought out. In 1916 he presented a case before this Society in which the picture was about the same as that described by Dr. Spiller. It was a case of a young girl with a right-sided hemiplegia in association with jacksonian epilepsy limited to the upper extremity. At that time he noted the features of sympathetic disturbance and looked up the subject and found the same factors that Dr. Spiller mentioned. He had been interested to see whether there was any representation in the cerebrum and exactly what part it had played in this picture, and found that the experimenters had been able to trace the sympathetic pathways only as far as the midbrain. Dr. Spiller asked Dr. Leopold to look up this report of his case and to find out whether it had been reported. He found that it had been reported only by title.

Dr. Charles K. Mills said that in connection with the discussion of Dr. Spiller's paper it was worth while to consider the relations of the basal ganglia, and especially the caudate nucleus, to the autonomic nervous system. In 1914, Dr. Mills contributed a paper on "Emotional Expression, Muscle Tonicity," etc., the main case reported in this paper being one in which Dr. Spiller made the microscopic investigations. The lesion was a markedly destructive one of the lenticular and caudate nuclei, the latter having almost disappeared. There was some evidence of loss of control over the autonomic nervous system in this case.

A CASE OF CEREBELLAR ABSCESS. Presented by Dr. Alfred Gordon.

L. G., a boy, aged 14, came under Dr. Gordon's observation because of an unsteady gait with a tendency to fall toward the right side. Examination revealed a purulent discharge from the right ear of several months' standing. The mastoid process was somewhat tender to touch. A history of otitis media following a protracted course of scarlet fever, preceded by diphtheria, was given. The boy's temperature was then 97.5 F. A blood examination revealed a fairly pronounced leukocytosis, namely, 17,800; hemoglobin, 60 per cent.; red cells, 3,420,000. The spinal fluid removed was bloody, and smears showed pus cells.

The interesting diagnostic symptoms were: Nystagmus was present. It was persistent, its quick movement was directed toward the left side. Standing on the right leg was difficult, but it was easy on the left. There was no Romberg sign. In walking there was a tendency to go toward the right side, and the head and trunk were also slightly inclined to the same side. When the patient, lying on his back, attempted to sit up, he raised his right leg first, while the left remained on the bed. On the other hand, when seated on a chair he was told to raise his right leg, he first flexed the thigh on the pelvis and then only succeeded in elevating the leg; in other words, hemiasynergia was present. In the well-known test for the finger to nose movement, hypermetria was evident in the right hand. It was also observed in the right leg as in each attempt to advance in walking he raised his right

foot off the floor higher than normally. Adiadokocinesis, as well as Holmes' and Steward's tests for abnormal voluntary and passive movements, were absent. The knee jerk on the right side was diminished when compared with that of the left side. The toe-phenomenon was absent with all the tests.

The most interesting peculiarity was seen in the past pointing test. With eyes open and facing the writer the patient was told to hold his right index finger in contact with that of the writer. He was then told to close his eyes, lower his arm and again bring the finger in contact to the former position. It was observed that the patient's finger would invariably deviate outward from the examiner's finger. A similar test with the left hand gave a normal result.

The patient's eyes, in addition to the nystagmus, showed a mild degree of papillitis on both sides. The pupils were normal. The ocular muscles were not involved. There was no other cranial nerve involvement. Sensibility of the face was not altered. There was no evidence of paralysis on either side of the body.

The patient complained of frequent vertigo and of constant though mild headache. There was no vomiting during the time of the writer's observation, although the patient formerly had not infrequent attacks of vomiting. Insomnia was persistent. The patient complained of shortness of breath. On examination it was observed that there was a persistent bradycardia. The pulse was 50. The mentality was not impaired.

In making a diagnosis the following possibilities were considered: temporosphenoidal abscess, labyrinthitis and abscess of the cerebellum. The history of otitis media with persistent otorrhea led the aurist to the consideration of a mastoiditis; but the symptoms enumerated above suggested a deep seated abscess in the area of the cerebellum. First, in labyrinthine diseases in which the vestibular apparatus is involved and in which there are also some symptoms common to cerebellar diseases, there are nevertheless present some manifestations which were absent in the latter, namely, Romberg sign; vestibular ataxia, which is, properly speaking, a static ataxia in which individual movements of the limbs are not modified while they are greatly disturbed in cerebellar cases; finally nystagmus usually had a tendency to disappear and it was always directed toward the sound side, while in cerebellar diseases it is constant. (The Bárány caloric test, which is negative in labyrinthitis, could unfortunately not be carried out in this case.)

In cerebral abscess of the temporosphenoidal region nystagmus is exceedingly rare. Equilibrium may be disturbed but it is not of the unilateral character of a cerebellar condition. Asynergia is absent. There is usually some degree of aphasia or paraphasia. There is also deep seated tenderness of the head on the side of the abscess. Mentality is always dull.

In the present case, on account of the special character of the nystagmus, of the hemiasynergy, of the past-pointing, of the dysmetria, strong presumptions were in favor of a cerebellar abscess on the right side, despite the fact that the mastoid process was tender. Accordingly an operation was urged over the cerebellar area, but the aurist insisted on a mastoid operation. A mastoidectomy was performed by him, but no pus was found and no necrotic bone was discovered. He then trephined the temporal region of the right side, but again no pus was found over the temporosphenoid area of the brain. The incision was then closed. The patient's condition became more and more

alarming, and he died on the next day. At necropsy a large abscess was found in the right cerebellar hemisphere. The pus contained streptococci (in large chains) and staphylococci.

The case is instructive from the standpoint of the diagnosis as one may be misled by the tenderness of the mastoid bone and consider only a mastoiditis. Necessity is indicated for a careful analysis of symptoms that may be observed in cases of otitis media thus leading to an accurate localization of the disorder either in the labyrinth, in the temporosphenoidal region or in the cerebellum. Exact surgical intervention is dependent on the latter.

ASYNCHRONICITY OF THE DIADOKOCINESIS FROM A MESENCEPHALIC LESION IN THE COURSE OF ENCEPHALITIS LETHARGICA. Presented by Dr. Tom A. Williams.

A woman was seen at Rutherfordton, N. C., with Dr. Henry Norris and Dr. J. C. Twitty, in September, 1919. In March, torpor and diplopia ushered in the attack, the acute phase of which lasted a month. Toward the end of May, hemiplegia occurred accompanied by dysarthria. These conditions had been gradually subsiding, but diplopia persisted except when the gaze was directed laterally. It was due to an insufficiency of convergence and more especially to the weakening of the left internal rectus. When the patient looked downward, however, fusion was possible. The reflexes were increased more particularly in the right lower limb, but both plantar responses were flexor. There was no change in the abdominal cutaneous reflexes. The gait was hesitating and somewhat swaying, and toward the right, increased with the intentional compensation exerted by the patient. The motility was only slightly weakened on the right side. There was no impairment of sensibility even the sense of attitude, and stereognosis being intact. Involvement of the cerebellar system was clearly demonstrated by the disturbances of the patient's balance when the left arm was violently thrown back, and by greater flaccidity in the left arm and wrist and by the greater number of the oscillations of the leg suspended over the side of the bed when the patellar tendon was tapped. The number of oscillations on the right side was five and on the left side seven, before the leg came to rest. These were all common enough differences where there was interference with the efferent cerebellar impulses. They were merely mentioned in order to give a perspective as to the condition present, which made the case worthy of report. The further fact was noted that while on the right side the pronator supinator diadokocinesia was normally rapid, 7.8 per second, the diadokocinesia on the left side, although also of even rhythm, was of greatly reduced rate, about 5.4 per second.

TO ILLUSTRATE THE METHOD OF REMOVING PHOBIA. AGAROPHOBIA COMBINED WITH CLAUSTROPHOBIA. Presented by Dr. Tom A. Williams.

A woman, aged 33, colored, had for eight years been unable to cross a wide street or to remain in a church or theater without intense emotional disturbance manifesting itself as palpitation, polypnea, pallor, chilliness, moisture and cyanosis of the extremities, rigidity and pain in the neck and back, nausea and sensations of weakness and dizziness. Examination showed no physical disease, other than mild hyperthyroidism.

The first occasion on which she had experienced similar sensations had been on a hot summer day, in a poorly ventilated church, when she had felt an overwhelming sense of illness. The compulsion to leave the church had been

intense, but because she was seated far up in front and did not wish to create a disturbance she had forced herself to remain. Some weeks later another attack occurred. They gradually occurred more often, but were at first avoidable by sitting near the door. Four years later, because of the sensation of oppressive heaviness which she feared might lead to unconsciousness, she had become afraid to cross a wide space. She had been an exceptionally strong girl, without worries, except anxiety over the health of an invalid mother. She had always feared high places, but thought nothing of it as the other members of her family all felt the same. There was no fear of the dark, no social timidities, other than dislike of being conspicuous as in class recitations.

The diagnosis was that the agarophobia and claustrophobia were hysterical in origin, arising from the powerful suggestion of the recollection of a particular experience, made efficacious now only by the timorous imagination of the patient. Relief of the chronic emotional strain was believed sufficient to cure this. Reeducation was forthwith begun. The patient was unwilling to introspect at first, but yielded when it was explained that to know one's self was as important a part of living as the understanding of technic is necessary to piano playing. It was explained that her dread of a close or open place was due to her ignorance of the mechanism of the consequences of a wrong way of looking at things and the consequent emotions, and that without a true insight into her own psychologic machinery she would be unable to control it. The power of induced ideas was illustrated by the story of the lawyer made sick by a few skilfully implanted ideas, as given in the play, the "Harvest Moon." After a while she accepted this explanation adding that "it must have been fear, for on the first occasion, when leaving the church, one of the maids said: 'What frightened you?'" In the course of a few days she wrote an account of her view of her own psychology in which she showed a clear grasp of the power of conceived ideas, and concluded by saying: "It is difficult for me to understand that these signs of illness are not illness at all but are caused by an induced impression. But now with the assurance that there is absolutely nothing wrong with my physical makeup, my problem seems to be to rid my mind of the fear which has unconsciously but completely controlled my thought."

The next step in the treatment was to demonstrate the truth of this. Dr. Williams accompanied her to a large square and made her cross it alone. Though evidently accomplished under great strain, she declared that she had performed the feat better than at any time since the beginning of the trouble. Later she attended church with only momentary discomfort. Dr. Williams concluded with the final adjuration, that now since she realized she was well all depended on herself.

This violent and persistent long continued agaraphobia and claustrophobia were traced to a single incident on which they were dependent. They were removed in less than a week by efforts directed toward giving the patient an understanding of their mechanism, indeed, compelling her to grasp it and then compelling her to take an exercise which afforded a practical demonstration.

Dr. Alfred Gordon said that in speaking of psychoneuroses, Dr. Williams made the remark that in these cases there was a special makeup of the individual. To Dr. Gordon's mind all psychoneurotics have a special makeup. All of us now and then meet with accidents and frights, and still we do not develop obsessions, but those who do develop obsessions always reveal a special makeup. To say that we can cure phobias or obsessions at one seance,

as Dr. Williams had expressed himself, Dr. Gordon could not see for the reasons mentioned how this could be done. Dr. Gordon thought all of the members of this Society had had a great many cases of this character. Some practice suggestion and find that they have the greatest difficulty in removing obsessions and other phenomena of the same nature. Others practice persuasion and still others psychanalysis. In the latter, Dr. Gordon does not always see the sexual element predominating. Dr. Williams practiced analysis of the people in the case he described. He tried to find the psychogenetic element in the case. He made the patient see for herself, he demonstrated to her the cause of her disturbance, and after the patient perceived the fault the trouble was removed. Dr. Gordon calls it persuasion. In regard to the difference in the results from persuasion and suggestion, suggestion was practiced from time immemorial. The speaker believed that suggestion alone in the majority of cases was of no value. In the method Dr. Williams described the patient particpated in the treatment. He laid before the patient the possible causes of the disturbance, and the patient seeing the mechanism, gradually improved.

Dr. W. G. Spiller asked, What was the essential difference between the method as described by Dr. Williams and the method of Dubois in treating the psychoneuroses? There must be psychanalysis in each method.

Dr. W. W. Hawke said that about three years before Dr. Patrick read a most extraordinary paper on this subject under the title "On the Obsessions of Fear," in which he went over this in practically the identical manner Dr. Williams had done. Dr. Hawke said he thought that practically every man had large numbers of these cases. He knew he had. He thought by knowing your case you could select the treatment.

Dr. Tom A. Williams said the method was not in any way especially different from that of Dubois. Dubois tried rational persuasion, but he did not in most of his cases endeavor to reach the groundwork, or mechanism, of the condition and the method was largely one of persuasion. Dr. Williams claimed his method was one in which the personality of the physician had nothing to do with it whatever, except that it was the personality which had taken the trouble to acquire a certain amount of skill in that particular sphere of therapeutics. It was a very difficult thing to do. If it were easy, we should all be doing it. It was done by a great many men in war; some acquired great skill in it. Some men would send back 50 per cent. of neuroses, some 90 and some 100 per cent. and a great many would cure the soldier in one sitting. The phobia is not cured in one sitting, but we give the patient sufficient material so that he can cure himself. It was necessary to go over the laborious processes of so-called psychanalysis in order to cure these patients. Dr. Williams said he had heard Dr. Patrick's paper and discussed it. Dr. Patrick had spoken very well about the fear psychosis, but he did not have success in curing the patient because he failed to cure a great many of them. Dr. Gordon hit the nail on the head when he said that suggestion was a very bad word. As soon as the patient understood the meaning of the fear, he was cured.

DOUBLING OF THE SPINAL CORD WITH PRESENTATION OF SPECIMEN. Presented by Dr. B. Lipshutz.

This paper will be published in full at a later date.

DISCUSSION

Dr. Alfred Gordon said he wished to call attention to a specimen he had exhibited nine years ago before the pathological society. In that case he had diplomyelia and in addition to it he found other conditions, namely, a true porencephaly in the right temporosphenoidal region. He found that the additional cord presented great variations in regard to the size of the cornua, and to the number of cells in each cornu. The original cord presented also malformations in regard to the number of cells, and to the shape of the cornua. The important thing to know was: In Dr. Gordon's case the boy, who was 15 years of age, was perfectly well up to that time and then began to develop epileptic seizures. It is interesting to know that congenital anatomic disorders are more tolerable and produce less disturbance during life than acquired pathologic conditions.

Dr. J. Hendrie Lloyd said that some years ago he had prepared a note on malformations of the spinal cord. It seemed to him that there might be some misunderstanding of terms, but he only looked hastily at Dr. Lipshutz' specimen. It seemed to Dr. Lloyd that this was a bifurcated cord, not a double cord. He thought a double cord was hard to imagine from an embryologic standpoint. Each of these branches is apparently half a cord. Some years ago there were some cases reported of so-called doubling of the spinal cord. He remembered seeing pictures of sections of two cords that were represented alongside of each other. Van Gieson of New York in 1890 published a paper in which he showed that these cases of so-called doubling of the cord were really artefacts, but Dr. Lipshutz' case was entirely different from these, for it seemed to show a branching of bifurcation of a single cord. Of course, the commonest malformation of the cord is that which is seen in spina bifida, and the query arises whether Dr. Lipshutz' case is allied to this malformation. A double monster, in which there is a true union of two distinct cords, must be rare; and still more rare must be a single embryo in which are developed two spinal cords.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, Dec. 18, 1919

George A. Waterman, M.D., *President*

PEARLY TUMORS OF THE BRAIN. Presented by Dr. Percival Bailey.

After some preliminary remarks on the occurrence and pathology of pearly tumors of the brain, Dr. Bailey presented a case in which the tumor, located in the fourth ventricle, was extirpated, followed by complete recovery.

DISCUSSION

Dr. H. C. Solomon: Among our routine necropsy examinations in the A. E. F. we performed one on a young soldier who had been killed by a gunshot wound in the hip which had become septic. On taking off the calvarium we were very much surprised to see a tumor mass which had eroded the skull. There was a definite depression in the brain and the membranes were torn. We thought at first that it was an endothelioma. It was adherent to the skull. When we examined it histologically it appeared to be a cholesteatoma. This was not only my conviction, but it was also the opinion of the other pathologists.

So far as known, there had been no symptoms and as he had been killed by a wound received at the front, it is to be presumed that he was normal in every way.

DR. HARVEY CUSHING: I would like to say a word about these pearly tumors. In my brain tumor series there are about 500 tumors that have been verified histologically. The two examples of these pearly tumors described by Dr. Bailey are the first that we have seen. Dr. Bailey has been through the literature very thoroughly and he finds only seven cases. These, however, present exactly the same kind of tumors that he has described. They are very different from the "cholesteatomata" mentioned by Dr. Solomon, which are not particularly rare. There are no cholesterin crystals in these pearly tumors. They are extremely rare, the percentage of incidence being something less than 0.2 per cent. in an ordinary series. Thus the two which have appeared in our series represent about the percentage in which they should occur. As in the tradition of all clinics, these two rare cases have occurred in conjunction.

In general, by far the most encouraging cases are the cerebellar cases, for they may recover completely. The acoustic tumors do not recover so well because there is always a residuum left. Most favorable are the cases of gliomatous cyst. When the cysts are thoroughly evacuated, if well treated, the patient may go on for years. I have now a good many patients with cerebellar cysts, a number have had repeated operations but a considerable number of them in our series are doing well, working and earning their livelihood, ten years after treatment.

A case of cerebellar tumor, I think, has a better outlook than a case of tumor in any other part of the brain. Of course the cerebellum is less vital in many ways, and the diagnosis is usually fairly simple. In the last nine months we have had about twenty-five cases; probably 50 per cent. showed no nystagmus. In the case presented by Dr. Bailey we surmised that the tumor was in the cerebellum, and we found that the patient had an occasional slight nystagmus, but if she had not had other symptoms we would have disregarded it. A great many of these cases have a median cyst, and with a median cyst there is no nystagmus. If the cyst is median, there may be some static instability but incoordination of movement of the upper extremities may be absent.

The most difficult point in diagnosis is to determine whether the tumor is frontal or cerebellar. It has interested me in looking over the report of Horsley's cases in the National Hospital to find in how many cases a frontal operation had been done and postmortem a cerebellar tumor found and vice versa.

BABINSKI'S THEORY OF HYSTERIA. Presented by DR. MORTON PRINCE.

The reader began by reciting the fact that of recent years, and particularly as a result of the experiences in this war, there has developed amongst French neurologists, under the teachings of Babinski, a reaction against the classical conception of hysteria of Charcot and his school. The present tendency, or rather the concept which is in vogue, is to regard the classical symptoms such as paralyses, anesthesias, convulsive seizures, etc., as artificially manufactured by the physician or the environment through the influence of suggestion and not as essential manifestations. In this view these maufactured symptoms are identified with hysteria and consequently hysteria becomes nothing more nor less than a group of suggested symptoms. There was evidence that Babinski's teachings were having considerable influence with British neurologists who

were engaged in the treatment of war psychoses in the army hospitals. The reader, therefore, undertook an examination of Babinski's views.

Babinski ascribes to hysteria only those "accidents" which have "the common characteristics of being capable of being reproduced experimentally by suggestion" which he avers is "capable of determining the form, the intensity and the duration of them"; and, correspondingly, they can be made to disappear by the influence of persuasion and suggestion. In these accidents or symptoms are included convulsive attacks, paralyses, various contractures, tremors, choreic movements, sometimes irregular but generally rhythmic, troubles of phonation, of respiration, or sensibility (anesthesias, hyperesthesias), and sensorial troubles.

It will be noted that the common mental symptoms, like amnesias, hallucinations, confusions, etc., have no place in this classification. From his point of view, Babinski would abandon the name of hysteria and replace it with the term pithiatism, from the Greek words meaning "I persuade" and "curable," which expresses, in his view, the fundamental character of these "accidents," namely, possibility of curing them through persuasion. Accordingly, he defines hysteria as "a pathological state manifesting itself through troubles which it is possible to reproduce by suggestion in certain subjects with a perfect exactness and which are susceptible of disappearing under the influence of persuasion (contra suggestion) alone."

This conception of a pathologic state, of which the so-called troubles are only manifestations, is a sound one: but after the formulation of this definition we hear nothing more of the pathologic state—only an exposition of its manifestations. This failure to keep in mind, on the part of Babinski, the conception of the pathological state, is the weak point in the edifice which he builds up. Dwelling only on symptoms he fails to grasp the essential problem of hysteria, losing sight of the pathologic state which should be the goal or solution sought. He confines himself to certain physiologic phenomena and loses sight of the fact that many of the so-called troubles which are the manifestations of the "hysterical state" are pure mental stigmas. These are not included in his group of pithiatic symptoms, i. e., are not recognized as hysterical, though they can be induced in favorable subjects by suggestion. Necessarily, therefore, he fails to recognize that many of the cases which he makes use of to support his thesis were already in the "hysterical state" and manifested classical manifestations of hysteria. These cases, he supposes, though having been exposed to emotional trauma, were free from hysteria until they were later the victims of suggestion. The consequence of this lack of vision is that when he comes to his data, to show that hysteria is not induced by emotion but by suggestion, he naively cites cases which every clinician ought to recognize had already developed hysteria and already exhibited classical hysterical symptoms of a mental kind induced by emotional trauma, or mental stress and strain, before suggestion, even in his own opinion, had got in its work.

The reader had no cause to quarrel with Babinski for insisting that hysterical phenomena can be suggested. Every neurologist has seen often enough examples of this, and indeed is aware that he himself has suggested such phenomena, intentionally or unintentionally. Nor can any one doubt that a large proportion of hysterical stigmas, particularly those following traumatisms, have been so suggested, generally unconsciously, by the examining physician. But that all have been so suggested, or what proportion have been so suggested, is another question. Given a certain state of mind, and symptoms can be manu-

factured almost ad libitum. In such an abnormal state the symptoms can be educated just as by reeducation they can be made to disappear. But what is the state of mind? That is the problem.

You cannot take any man in the street, or even any patient with organic nervous disease, such as tabes, and by suggestion create hysterical stigmas. What is the difference between the normal mental state of the man in the street and that of the person who has undergone some sort of psychologic conflict, or trauma, or shock, that the one is practically immune to hysterical stigmas by suggestion and the other permits them to be created with ease? This is the crux of the problem. As a matter of fact, the hysterical state is a condition of mental dissociation accompanied by certain automatisms due to psychologic factors. The real point, therefore, is whether this state can only occur by the force of a direct suggestion or whether it may be the result of other forces, such as the discharge of an emotion, or by the repressing or inhibited force of other psychologic factors involving a complicated internal mechanism, etc. Babinski rejects emotion as a possible exciting cause and finds that suggestion is the only possible cause left. As one of the links in the chain of his argument he contends that hysterical phenomena never appear at the moment of, or immediately after the emotional shock, when the emotion is at its height, but that always, "between the emotional shock and the presence of hysterical (pithiatic) accidents there is an intervening phase, sometimes quite long, which Charcot called the 'phase of mediation,' during which autosuggestion or heterosuggestion have the opportunity to intervene" and induce the accidents. In support of this view he cites the observations of numerous writers who had the opportunity in this war to observe so-called "shell shock" at the front and behind the lines. Babinski seems at first to be supported by these observations, for it seems that those symptoms of hysteria which Babinski elects to call alone pithiatic or hysterical, that is the paralyses, anesthesias, etc., rarely developed at the time of the emotional shock but appeared after an interval, when the soldier had reached some place behind the lines.

A study of the reports, however, shows that the patients, apparently without exception, exhibited at the emotional period immediately following the shock, hysterical mental symptoms of a very marked character. Among the symptoms commonly described were amnesias, hallucinations, deliria, inability to respond to questions, even when attempts were made to force a response, apparent incapacity to perform a voluntary act, states of hebetude, stupor, confusional states, states allied to fugues, mental dullness, irrational states, tachycardia, tachypnea and tremor.

What are these, it may be asked, with the exception of the three last (which are only the physiologic manifestations of emotion) but states of dissociation with automatisms or unregulated and uncontrolled functioning of disintegrated psychologic systems? They are from the modern point of view typical and pure symptoms of the hysterical pathologic state.

That certain symptoms like paralysis, anesthesia, dumbness, deafness, etc., do not appear until a later period may be a fact, as a matter of observation, but the real question is *why* do these particular symptoms appear only later, while other and mental symptoms appear at the height of the emotional discharge? It is a question of the WHY. The fact which seems to have been brought out by observations during this war is that the hysterical state manifesting itself by mental dissociation, can be induced immediately under

mental stress and strain at the moment of the emotional discharge, while certain other symptoms, in the great majority of cases, develop only after a period of incubation.

Babinski's fundamental error is not recognizing that the hysterical state is one of functional dissociation and that any psychologic factor capable of producing such a dissociation, whether it be emotion or a conflict, is capable of producing hysteria. The mechanism by which individual symptoms is produced is another problem. It may be suggestion, as we all know, or it may be a very complicated mechanism which still requires solution.

DISCUSSION

DR. E. W. TAYLOR: I presume nearly everyone would agree with Dr. Prince as against Babinski's attitude. It seems to me to be clear, just as Dr Prince has said, that for many years the fundamental idea of psychologic mechanisms have been coming into prominence which lie behind the physiologic symptoms which Babinski has described. In other words, Babinski described, whereas others have done rather more, to interpret.

DR. WALTER B. SWIFT: I agree with the thesis of Dr. Prince in almost every respect, if by "disassociation" he means an entirely conscious process. I would like, however, to say a word in favor of Dr. Babinski. The background of his original training and his experience has not been chiefly psychologic. As he is principally an internist, his medical attitudes being therefore chiefly observational, he is primarily an observationalist. We would, therefore, naturally expect him to describe observations rather than to create or formulate psychologic concepts of hysteria. He illustrated this attitude clearly when I was in Paris and saw him demonstrate his reflex. He showed three actions besides the extension of the great toe which constituted that reflex. I was more interested in his acuteness of observation than I was in the details of that reflex. Again, how well his attitude of mind is illustrated by his holding, some years ago, that hysteria constituted the symptoms; or, in other words, that the symptoms found in hysteria are the disease.

Further researches are needed today on hysteria. This is a great field still unexplored. Some of you men with a large hysterical clientele in your clinics should take these cases through the steps that I have taken stammering. We medical men first interpreted stammering as being a physical thing; later a vocal diagnosis was made. Finally the diagnosis was traced to the nervous system, until in the end the defect was found to be lodged in the subconscious mentality. At this time I entered the field and discovered the conscious mentality of it. I found a constant conscious psychologic lesion. It was a vitiation of visual processes. The field of hysteria today is ripe for a similar contribution. The conscious mentality of hysteria, decidedly, needs full investigation. With our new modern psychologic tests and recent new medical avenues for conscious mental investigation (found in speech tests) we ought to enter the field of the conscious mentality of hysteria and discover something new, perhaps the real final lesion.

I was interested to hear Dr. Prince present Babinski's interpretation of hysteria as the externalization of will-processes. To me this interpretation is entirely unsatisfactory, if he means by will-processes the conscious choice that has been taught us by James. Zehien once demonstrated in the Charitè Hospital a case of hysteria that simulated tabes, and another case of hysteria that simulated multiple sclerosis. That these two cases could be nothing more than externalization of will-processes is hard even to imagine. I consider

hysterical manifestations as the externalizations of a conscious lack—an absence in the conscious mentality, which lets hysterical phenomena appear in a similar fashion as the lack of the central motor neuron lets the pathologic reflexes and spasticity appear. A description of this conscious lack is what we need in hysteria today.

FRACTURES OF THE SPINE. Presented by Dr. W. J. Mixter.

The most important group of fractures of the spine are those with cord involvement, and this paper deals largely with them. Treatment must depend almost entirely on the cord lesion except in so far as the bony lesion has bearing on the cord lesion. Laminectomy with an attempt to remove the cause of injury to the cord or to alleviate the effect of such injury is, of course, the operation usually performed. In view of the pathologic considerations early operation would be indicated in these cases: (1) Partial section of the cord, (2) marked contusion or edema of the cord, (3) pressure on the cord from any cause, (4) bony injury which is likely to cause cord injury in the future, (5) severe cases of hematomyelia. Operation would be contra-indicated in the following cases: (1) Complete section of the cord, (2) moderate angulation without pressure, (3) mild edema of the cord, (4) concussion of the cord, (5) moderate hematomyelia.

Splitting the cord according to the technic proposed by Allen may be performed in certain cases, but without great hope of a successful result.

Early operation is most important and if possible should be performed within twenty-four hours if the symptomatology seems to point to any of the pathologic changes noted above as requiring operation, the most important single indication being increase of symptoms.

The care of the paralyzed patient, whether with or without laminectomy, is difficult and the most careful nursing is essential. The lessons of the war are important and the early establishment of an "automatic" bladder most desirable. The Balkan frame is often of help although as a rule a water bed is sufficient. A plaster jacket is unnecessary in most cases except where the fracture is in the cervical region.

If we are to be at all successful in this most discouraging type of surgery we must make an early complete examination of the acute case. If after careful review of the findings operation seems indicated and the patient is not in severe shock, let the operation be performed at the earliest possible moment.

DISCUSSION

Dr. Harvey Cushing: This is a most difficult subject. I very heartily agree with most of what Dr. Mixter has said. I am glad that he is conservative about these cases. I think most people are who have had a great deal to do with them. The war brought out many interesting things about spinal injuries. The mortality among the cases was very high. The first large group of cases that I saw was during a period of service with the British, particularly after coming in contact with Holmes and Sargent who had been collecting these spinal cases. We must wait until they have had an opportunity to assemble their material and study it before we can have what they have promised to give us, a thorough analysis and study of a series of cases in which every single segment of the spinal cord is involved.

The most interesting cases were the lower cervical transverse lesions. The patients had amazingly low temperatures. Men lay in the wards, conscious,

comfortable and with complete anuria for a period ranging from four to five days with a temperature sometimes as low as 90 F. I saw two men, who had a high cervical transverse lesion, with a temperature in the eighties absolutely comfortable and taking nourishment.

Dr. Mixter has spoken of another very interesting group of patients, and to us who had the surgical care of these patients the question which Dr. Mixter has spoken of was brought up, namely, that of keeping them clean. The question of debate was "How shall the bladder be taken care of?" because infection is almost inevitable. I have a friend in the French army who is a genito-urinary specialist and has made a study of the bladder sphincters and their action. His best friend, an aviator, fell and broke his spine in the upper thoracic region. He took care of the patient and would not let any one else touch him. He catheterized him himself with the greatest possible care. The boy died inside of three weeks of an acute surgical kidney. With every possible precaution these infections may occur. A catheter drainage means an infection inevitably. Should we leave these patients absolutely alone and let the bladder distend until it dribbles, a method very strongly advocated by the late Dr. Murphy of Chicago, or should we immediately do a suprapubic operation? One can make a puncture over the pubis with a split trocar so that the tube is introduced with no leaking. The method of leaving the bladder alone and letting it distend was adopted and official regulations were made that such patients should never be catheterized. However, in most instances, some one would be on the ground who would fear to let the bladder distend sufficiently and catheterization would be inaugurated. This particular point has never been satisfactorily settled but the thing behind it of particular interest to me is the fact that the spinal cases, cared for in Dr. Head's special hospital, were patients who had escaped bed sores and cystitis. If they escaped from an infection, these automatic states that Dr. Mixter has spoken of usually began to appear. Some of these patients learned just what stimulus they needed to discharge their own bladder.

Of interest from the pathologic point of view are the observations that Mme. Dejerine has made of the spinal cases which have been under her care at Les Invalides. I saw there a great many roentgen-ray plates of her patients and many of these had begun to show bony deposits in the paralyzed muscle.

I do not know how any more definite rules for operation can be laid down than those Dr. Mixter has given. Each case is a law unto itself. It is difficult to foretell what the injury is. I think we have been misled by the fortunate results of operation in which the recovery has not been attributable to the operation. A laminectomy is quite a difficult procedure. The patients are not in the best possible condition for an operation. If the spinal cord were as accessible as the brain, we should feel much more inclined to operate for contusions. If the brain is treated surgically and the contused area removed by operation, it will recover much more rapidly. If we could get at the cord easily and clean out its area of contusion without, of course, damaging it and could do this quickly, by an operation which was not an operation of magnitude, we would have conditions under which we might wish to operate more often. I find that in the more encouraging cases we are rather apt to refrain from operating for many of them do well. In the more discouraging cases we are also rather apt to refrain from operating for they do badly in either case. This is a general rule to which there are, of course, exceptions.

DR. WILLIAM J. BRICKLEY: I think Dr. Mixter is absolutely right in saying hold back until you are sure and operate as soon as you can be sure. These cases as I have seen them change from hour to hour. A case of broken back is plus shock, concussion and great laceration. You wouldn't be justified in operating when the picture is that of a blow across the back, you usually have internal injury as well—for example, kidneys or intestine ruptured. Two or three times I have seen ruptured bladder with broken back. That, perhaps, accounts for some of the urinary difficulty following a case of injured cord. It will be well, therefore, as I see it, not to make a diagnosis on one visit but perhaps in two hours to examine the patient again. As I have seen the necropsies in some of these cases I have been surprised to see how small an injury from the pathologic standpoint it takes to kill a person. In other cases it is surprising to see how much movement we have with a badly damaged cord. In other words, begin at once to treat your patient. Do not forget that there is always something else besides a broken back, if one can discover a broken back readily with the fingers. Usually the other injuries will have to be treated first. Get rid of the shock, investigate the ribs, the urinary tract and intestinal tract, before you operate for if you do not the best operation will not be of avail. In civil practice we cannot just say operate, as is often possible in a hospital. When you are dealing with general practice people often simply will not permit you to do the thing which offers the best chance for the patient. They have to be convinced. The average man thinks that laminectomy is difficult. Therefore he opposes many such operations. I have been suprised in looking over a good many cases in industrial work to see what good recoveries some apparently complete paralytics have made. In two or three years they are working. We do not operate often enough on this type of case. Dr. Cushing expressed the sentiment when he said certain cases are so difficult that we do not feel like taking the risk. I was interested to look up this matter because I have had many cases of injured backs that I have had to pass on, at one time approximately twenty or thirty cases a day, with the idea of finding out why they could not go back to work, and it was discovered that many patients that were being treated for sprained back or lumbago actually had broken backs of some degree or other. When a person does not get well and has a persisting train of symptoms, it is just as well to take a roentgenogram.

DR. MORTON PRINCE: I was intensely interested in hearing Dr. Mixter and also Dr. Cushing and Dr. Brickley because I wanted to learn what the most recent modern experience has been, and what are the conclusions which, in particular, the surgeons of this war have drawn from their experiences. I wanted to see what change had taken place in the point of view. I have been much surprised by the similarity of views expressed here to those that were held in the discussions that took place twenty years or so ago. This whole discussion sounds, in fact, like an echo of similar discussions on fractures of the spine which we used to have then. I, personally, have not had an experience comparable to that of those who have given their views tonight, but in the course of some twenty-five or thirty years' service at the City Hospital, I have seen a good many cases and have taken the responsibility of advising operation in a good many. For a number of years I was intensely interested in the subject. I recall that the question of the advisability of operating has come and gone in waves. Following a time of indifference, there came a great wave of interest accompanied by the hopefulness and fervor for operating. That was just after Horsley brought out his operations

on the brain. For a time this wave of hopefulness continued, and then it was followed by discouragement and pessimism. Then another group of younger surgeons came along and this group became enthusiastic, took up the subject again, and operated but with the same results. And so it has continued. I confess, as a result of my experience, I have been left with a feeling of absolute pessimism.

I cannot see from what has been said here that anything has been gained by the experiences of the war to show that if the cord has been injured that anything can be done by operating to restore function. The reader's sole conclusion is that if we are going to operate, we should operate early. Of course, I do not understand that Dr. Cushing adds anything more optimistic or pessimistic.

Concerning the question of diagnosis, as to whether there is a complete or only a partial injury of the cord, we encountered the same difficulty during the years of which I spoke. It is certainly a very difficult matter to tell whether one has a complete or partial injury, and I cannot see that anything has been added to our diagnostic armamentarium. I am glad the point has been emphasized by Dr. Cushing and Dr. Brickley that there are a good many cases of apparently complete and partial injury which afterward have improved, and improved decidedly, without operation having been performed. Therefore it follows that if improvement follows operation it may in no way be due to the latter. It is always a question whether it is not better to give the patient a chance to improve without operating on him.

Dr. Walter B. Swift: It has been mentioned repeatedly that rapid or marked recovery has occurred in lesions of the cord. I would like to ask if there is any new evidence which can more directly than ever before trace these recoveries to the growth of new neurons in the cord, rather than to the recovery of old neurons which have been put out of function?

Dr. A. E. Brownrigg: In a case seen in consultation with Dr. Shea of Nashua six or eight years ago, I made the following observations: The patient had fallen from a building violently buckling his back with consequent fracture. Several laminae were removed. The dura was punctured and beneath the point of injury was a large area of fusiform swelling which proved to be blood clot. It appeared that three fourths of the cord was severed. The operation was finished and the patient was completely paralyzed below the level of the lesion. After several years there was some recurrence of sensation in the lower extremities and also some power of movement. He has since been able to earn something by selling small articles on the street.

Dr. Brownrigg expressed himself as optimistic that the operation had been of decided value in helping the cord to unite.

Dr. E. W. Taylor: As a matter of fact, I think there is no experimental evidence whatever that the cord itself recovers, that the neurons grow through. In those instances in which this has appeared to take place, presumably the regeneration takes place through the cord that remains.

Some photographs were shown illustrating the types of lesion that usually occur. One thing that has been very striking is the fact that there is almost never any subdural hemorrhage. As a matter of fact, traumatism to the spine leads to a central hemorrhage almost always and practically never to a subdural hemorrhage, possibly occasionally to an extradural one. Less conservatism in operation owing to improvement in technic is apparently the watchword of the future.

DR. W. J. MIXTER: I think we may say that in fractures of the spine with or without cord lesions there are very definite surgical indications. Certain lesions definitely call for surgical procedure with a distinct hope of improvement. The great difficulty at the present time is the question of diagnosis of these lesions. Allen's work is positive in regard to the benefit that may be obtained by splitting the cord, but one cannot tell in which case the cord ought to be split. That is one of our main reasons for pessimism at the present time. I agree with Dr. Taylor that a subdural hemorrhage is a rarity. The extradural hemorrhage seems more common, but neither of them are of frequent occurrence.